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IMMEDIATE REPAIR OF INJURIES OF FACE AND JAWS.*

EDGAR M. HOLMES, M.D.,
Boston, Massachusetts.

On admission to a hospital, any crash victim must be evaluated as a whole, whether the apparent injury is to the head, body or extremities. In facial injuries, an unobstructed airway is of first consideration and, except in mandibular, hyoid and thyroid fractures, can usually be obtained by sucking out blood and secretion. In the unconscious individual, the insertion of a mouth airway may be all that is indicated. Should there be collapse of the hyoid, thyroid or trachea, an anesthesia intubation tube will control the situation admirably until treatment may be undertaken.

By the time of admission, brisk bleeding from lacerations or from areas within the nose or sinuses due to fractures has usually stopped; but, if not, the simplest method should be employed to control it until complete evaluation of the patient has been possible and definitive treatment is outlined and undertaken under operating room conditions. It is better to rely upon blood replacement than to utilize postnasal or intranasal packing with its inherent risk of complications, though packing is occasionally imperative.

Should there be injuries other than those of the face which

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necessitate a delay in our treatment, the soft tissue wounds should be evaluated and treated as indicated. All gross contamination should be removed and the wound irrigated with volumes of sterile saline to remove any unseen contaminants. If the wounds do not communicate with fractures, they may then be closed, meticulous care being employed in reapproximating any severed, deep structures, followed by similar approximation of the cut skin. The parotid duct and facial nerve can be a challenge in restoring function. If there is grossly devitalized tissue or skin, this may be removed, particularly if it is subcutaneous. If there is a jagged scar or tangential cut which we know will leave an irregular scar on healing, the edges may be cut away so that the basal layers of the skin may be carefully approximated. Should this debridement be in an area which would result in subsequent distortion of contour, it is frequently advisable to approximate the ragged edges and, at a later time, when all sepsis and tissue reaction has abated, to perform a secondary improvement of the scars.

Whenever the lacerations are in continuity with the fracture site, I like to leave this area approximated by one or two superficial stitches or merely protected by a dressing to eliminate external contamination until the fractures can be reduced and immobilized. To suture the area carefully before reduction of the bones may necessitate the removal of all the stitches, before our reduction and fixation can be carried out, thus introducing an unnecessary procedure.

Having established an adequate airway and controlled bleeding, there is then no urgency to reduce facial fractures, though they should be reduced as soon as practical. This allows ample time for careful evaluation of the injured person. Physical and X-ray examination together with all necessary laboratory work enables one to institute treatment which will prepare the injured for surgery. Once the patient's general condition has been stabilized and he is a safe anesthesia risk, we may proceed with an organized plan for reduction and fixation of the displaced parts. If a long delay of days is obligatory, it permits organization, fibrosis and early

healing to begin which must be overcome before exact approximation can be obtained and the process of healing may resume.

In treating the fractures, our basic goal is to realign the broken bones and then immobilize them until healing results. Methods vary, but the simplest technique should be employed which will serve our purpose. Avoid elaborate, complicated appliances which, in themselves, could create complications.

NASAL AND SEPTAL FRACTURES.

The majority of nasal fractures involve the lower third of the nasal bones, and associated with them, the lower thin portion of the ascending processes of the superior maxilla. These simple fractures are readily reduced and fixed. When the blow has been received on the dorsum from before backward and there has been a posterior displacement of the nasal bones, there is invariably a fracture and dislocation of the cartilaginous septum and, very commonly, the perpendicular plate of the ethmoid as well. If the septal fractures can be brought into position and splintered, this automatically maintains a support to the nasal bones and cartilaginous elements. Failure to maintain this support, which is similar to a tent pole, results in a later contracture due to scar tissue and a depression of the dorsum. Whenever there is evidence of a disarticulation of the nasal bones from the frontal bone, particularly with posterior displacement, one must always assume there is a cerebro-spinal leak until proven otherwise. If there is a cerebro-spinal leak, it is the consensus that early reduction and fixation of the fracture is indicated. This is particularly true when it is associated with separation of the facial bones from the skull, because every time a patient swallows, the facial bones move, and with this movement, the dural tear is disturbed and the leak cannot possibly close. Once reduced and the bones fixed, this motion is eliminated, and the dural tear usually seals over without complication. The majority of nasal and septal fractures may be splintered by intranasal packing plus an external molded splint. It is always comforting to the patient if a fairly large catheter

is first introduced along the floor of the nose before packing is placed on top of it. In some of the extremely comminuted fractures, particularly where the ascending processes are still intact, the nasal bones and septum may be supported by through-and-through wire sutures applied over a soft metal plate. There are some comminuted, depressed nasal fractures which require support from an intranasal appliance attached to a headcap. This is especially true when one is dealing with multiple facial fractures.

ZYGOMATIC FRACTURES.

The zygoma is roughly a flat bone with three main articulations. The articulation with the frontal bone, the superior maxilla and the zygomatic arch of the temporal bone. The largest and firmest articulation is with the superior maxilla. If this articulation is separated, the maxillary antrum occasionally may be entered. The bone is usually displaced as a unit though comminution may occur and is displaced in the direction of the striking force. Its reduction should entail a force in the opposite direction. The bone forms the outer and lower third of the orbit so that the orbit may either be compressed, creating exophthalmus, or enlarged, resulting in an enophthalmus. If the floor of the orbit is torn, the orbital fat may protrude into the maxillary antrum creating an enophthalmus even though adequate reduction and fixation of the bone is obtained. The lateral palpebral ligament is attached to the frontal process so that when this suture is separated and moved in any direction, the function of the eye is altered with it. It is, therefore, imperative that all fractures in which this articulation is separated should be reduced and fixed.

There are four fundamental approaches for reducing the zygoma and the method which best fits the case should be employed.

When the zygomatic arch has been broken and depressed without displacement of the zygoma itself, the best approach is that which exposes the fascia of the temporal muscle just inside the hairline. The fascia over the area goes down and attaches to the zygomatic arch. By cutting through the fascia

so that a blunt elevator may be inserted beneath it, it will be directed by this facial plane beneath the depressed arch which may then be elevated. This method may also be employed for elevating some of the zygoma which has been rotated into the temporal fossa being hinged along the sutures of the frontal and maxillary bones. When there is a displacement of the bone posteriorly, however, a more direct force and leverage may be applied through an incision in the buccal mucosa opposite the last upper molar tooth. Through this incision, a blunt elevator may be inserted to the posterior surface of the bone. Occasionally, a combination of these two approaches is necessary if the bone is impacted. When the bone is pushed medially or posteriorly, collapsing the antrum, I like to use the intranasal approach. The naso-antral wall is removed beneath the inferior turbinate through which a specially designed hook with a large handle may be freely inserted. Its blunt tip is placed beneath the solid part of the zygoma and by palpation with one hand externally and pressure through instruments from within, the bone may be guided and directed into position. Bones which have not been freely mobilized by injury but are hinged, may frequently be reduced in this manner without fixation. If, however, the bone is freely floating, it is then imperative to fix the bone by direct wiring. If there is evidence that there is much comminution of the bone, particularly the floor of the orbit with loss of orbital support, it is then necessary to expose the front face of the antrum, remove the bone as in a Caldwell-Luc operation which permits inspection of the floor of the orbit. Direct approximation and wiring of the bone may then be made and if the floor of the orbit is sagging or orbital contents have protruded into the antrum, they may be replaced and supported by packing. Occasionally, the loss of support is so great that this does not seem sufficient, and a graft of a thin plate of bone may have to be inserted between the periosteum and the remaining floor of the orbit to retain the orbital contents.

MAXILLARY FRACTURES.

In mid-face fractures with displacement of the maxilla,

there is invariably a posterior displacement with premature occlusion of the teeth which results in an open bite. Fractures are frequently impacted so that considerable force must be exerted before they can be realigned. To unlock impactions, downward traction on the posterior bony palate by a heavy hook placed through the soft palate together with firm jaw forceps applied to the alveolus through which controlled rocking may be applied, usually suffices to free up the elements. There are some cases which may be better reduced by prolonged, constant, elastic traction between the teeth of the maxilla and mandible, or even through wires to arms protruding from a head cap. Once freed and brought into position, the simplest form of fixation should be used. If the zygomas are broken away from the frontal bone, they should first be wired directly. If there is another separation between the zygomas and the maxilla, direct open wiring may be utilized here. If there is still mobility of the maxilla, the component parts may be more firmly held together by a wire extending from the zygomatic process of the frontal bone down to a hole drilled in the maxilla or even brought down and wired to the teeth. This creates a suspension form of wiring which holds the bones together as though in a sling. Another method of support is the utilization of a modification of the Kingsley splint which attaches to the upper teeth and has arms which come out through the mouth and arch back over the cheeks. The external arms are attached to a headcap by means of elastic traction. Some surgeons employ Kirschner wires to establish fixation, but I feel these should be limited to the very few who are exceptionally trained in their use as I have seen unnecessary complications when used by the inexperienced.

MANDIBLE.

The mandible may be considered a "U-shaped" bone which is attached by its open ends by means of loose hinges; may be moved about ten degrees from side to side; may be pulled one-half inch forward but cannot be pushed backward because the ends are thrust into a solid socket. This bone will, therefore, give with many blows. Blows which cause the "U" to

break are those delivered somewhere along the bend of the "U". The break may be at the site of pressure or may occur distant to the blow on the opposite side. We may have one, two or more fractures with, or without, disarticulation of the joints. Occasionally, there will be a break at the symphysis with a lateral spreading of each condyle with disarticulation of one or the other, associated with a tear through the external auditory meatus. Rarely, the condyle is pushed up into the middle cranial fossa. When there has been a fracture of the mandible, there is almost always a disturbance of the bite associated with swelling and tenderness about the site of the break together with pain on motion. With this suspicious evidence, further manipulation, palpation and X-rays will usually confirm a break.

As brought out before, the simplest method should be employed for reduction and fixation of all fractures and when we have teeth, they may suffice for both. Our objective is good dental occlusion and function, and a firm union. There are all types of dental appliances which may be obtained and used for dental fixation, but heavy arch wire and 28 or 30 gauge stainless steel wire are usually sufficient for most reductions and fixations. In an edentulous individual, circumferential wiring about a denture and open reduction permitting inter-osseous wiring will hold the fragments in good position and permit early motion of the mandible. Whenever there is a tooth in a line of fracture and it is needed to maintain position of the bone, it should be retained. With adequate doses of antibiotics and good oral hygiene, secondary sepsis is held at a minimum and, should sepsis occur, the tooth may then be removed and, by that time, bony union may have started. If the tooth is fractured and has no function in maintaining reduction, it should be removed. When there is but one tooth on a posterior mandibular fragment, it is imperative to maintain it if possible, to control the vertical position of the proximal fragment until healing occurs. It is frequently advisable to perform an open reduction and wiring at the free border which will usually create exact approximation which is not always possible by tooth fixation alone.

It is rare in civilian accidents to have a segment of the

mandible completely destroyed as we have in projectile injuries, though we may have a section so comminuted with loss of teeth and separation of the proximal parts, that the ingenuity of the surgeon is taxed to re-establish good continuity, healing and function.

Assume there is a comminution of the mandible about the symphysis which spares the two sides. The two sides are brought into proper occlusion and splinted. The fragments of bone are then held in as near alignment as possible, either with, or without, open reduction until healing results. This prevents displacement of the main segments of the mandible by soft tissue contraction.

If there has been lost bone, the remaining bones should first be aligned with the upper jaw and held in position. Once soft tissue healing is complete and all infection has been eliminated, a rib or iliac bone graft may be inserted to restore the mandible. If the two side bones have not been maintained in proper alignment and have been permitted to be pulled together anteriorly, it is then much more difficult to eliminate the contracted scar tissue before inserting a graft.

Fractures of the condylar neck almost invariably result in a rotation of the head of the condyle by muscular pull of the pterygoids. If the fracture is on one side only, the general consensus is that a satisfactory functioning joint may be obtained by fixing the mandible in proper dental occlusion for about five weeks. At this time, the fusion or fixation of the head to the remaining neck has been obtained and a functioning joint will result. If the fracture is of both condylar necks, similar treatment may be used with a good, functioning result, though it cannot be assured as in the unilateral fracture. If there has been an anterior dislocation of the head and this cannot be manipulated back into the fossa, it then becomes necessary to perform an open reduction with wiring of the fracture. If there is a fracture of the coronoid process, which is rare, there is usually minimal displacement and unless there is reduction of mandibular function, there is no need for treatment.

SUMMARY.

In all facial injuries, the patient should be considered as a whole. Early emergency care should establish an adequate airway and control bleeding. Normal body physiology should be established before undertaking extensive repairs. Soft tissue lacerations should be cared for after bony reduction and fixation except in cases where delay in reduction is necessary. Early reduction of fractures is advisable, and the simplest form of fixation which will not introduce secondary complications should be employed.

330 Dartmouth Street.

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A continuous course of two weeks' duration is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and the Graduate School of Medicine of the University of Pennsylvania. Sessions will be held daily at the Temple University Medical Center from 9:00 a.m. to 5:00 p.m., March 5-16, 1962. Tuition Fee—$175.00. Enrollment limited. Dr. Louis Tuft is course director and Drs. George I. Blumstein and Merle M. Miller are associate directors.

The course is designed for physicians desirous of extending their knowledge of allergy. It could serve as an introductory course for those about to enter the field or as a review course for practicing allergists.

For brochure and application forms write to: Dr. George Blumstein, c/o Temple Medical Center, Philadelphia 40, Penna.

CONCEPTS IN FACIAL FORM AND FUNCTION REHABILITATION.*

JOHN J. CONLEY, M.D.,

New York, N. Y.

The concept of form and function in the rehabilitation of wounds in the head and neck is the essence of therapy in an era of speed and violence. Thirty-six per cent of the injuries to the body in accidents occur in the region of the face and neck, with their distressing possibilities of both esthetic mutilation and distortion of function.

Alterations in function in the head and neck area apply to the interference with the special sense end-organs, the region injured and the type of assault. Injuries to the bony and cartilaginous framework involved, in the order of frequency are: the nasal pyramid and airway passages of the nose. The interference here is essentially with breathing and an alteration to nasality of speech with a displacement or crushing injury to the nose and nasal septum. Chewing is affected by injuries to the mandible. Unilateral or bilateral blindness, diplopia and epiphora are often associated with trauma to and about the orbit. Injuries to the base of the skull with fracture may result in an interference with olfaction in the anterior fossa, sight, at the base of the middle fossa and equilibrium, in the posterior fossa. Fractures passing through the mastoid and petrous pyramid are often associated with deafness and vertigo. Fractures or crushing injuries to the larynx and trachea interfere with the functions of breathing and speaking.

Soft tissue injuries are characterized by lacerations with resultant scar formation involving the skin, muscle, organs, arteries, nerves or ducts. Contusions are usually associated

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with crushing injuries of the soft tissue involving the face and neck. Avulsions are characterized by a loss of superficial and deep soft tissue substance involving the scalp, ear, eye, lips, nose and surface skin. Burns are associated with heavy hypertrophied scar contraction and deformity affecting the skin, muscle and movement of the head and neck.

These defects are discovered by inspection, palpation and X-ray examination. Definitive therapy cannot be carried out at the scene of the accident. The injured are first seen by other motorists, police, mechanics and usually a general physician. The airway is established, hemorrhage is controlled, shock is treated and the patient is transported to a hospital. Here an advanced service inspects for asymmetry of the face; the characteristic depression of the malar compound or zygoma; the sunken eyeball with diplopia, or ecchymosis; the displaced, flattened nose with internal nasal obstruction; a floating maxilla and palate; malocclusion of the maxilla or mandible associated with trismus; a spinal fluid leak through the nose, mouth, or ear; nerve injury, with an absence of sensation in the supraorbital, infraorbital or alveolar areas or paralysis in the region of the VIIth cranial nerve; duct injury, hoarseness, and airway obstruction.

When the patient's general condition warrants intervention, a plan of repair is outlined. The sooner the repair is accomplished the better one might expect the results to be. A patent airway system during the operation and postoperatively is a fundamental essential. The anesthesia may be local or general. The soft tissue repair is always preceded by careful evaluation of the bony and cartilaginous status. The various methods of management of these deficiencies have been excellently outlined by Doctor Holmes.

The soft tissue repair consists of abundant irrigation of the wound, a careful removal of all foreign body debris, an absolute minimal amount of debridement and a removal of only the sections of bone that are free. The mucous membrane is repaired first, and then a careful repair of lacerated ducts, nerves and muscles in a three-dimensional fashion is accomplished. Soft tissues are sutured in layers with catgut. The

subcutaneous tissue is carefully approximated, the dermis is only slightly undermined in order to accomplish eversion and the skin approximated without tension with #000000 dermalon sutures.

When there is a loss of tissue there should be no hesitancy in dressing the wound with free skin grafts or the use of regional flaps.

There is a synergistic effect of taking into account in all injuries of the face and neck, the functional obligations of the injured region and its basic form. When these are dissociated the inevitable deficiencies are compounded. In the preparation of the wound, functional rehabilitation is established to a maximum degree. There is no substitute for restoration of normal anatomy when this is possible, beginning with bony and cartilaginous architecture and then proceeding to the other layers of anatomical structures. Establishment of basic architecture always facilitates contour restoration and minimizes the necessity for secondary repair.

Note: A series of slides demonstrating the use of rehabilitative techniques to re-establish form and function, followed this presentation.

139 East 36th Street.

THE MOUNT SINAI HOSPITAL.

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum, and Otoplasty will be given January 13, 1961, to January 26, 1962, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N. Y.

EVALUATION OF SUBTOTAL LARYNGECTOMY BASED UPON STUDIES OF THE LYMPHATICS OF THE LARYNX AND NECK.*†‡

JOEL J. PRESSMAN, M.D.,
and
MILDRED BURTZ SIMON, R.N.,
Los Angeles, Calif.

LYMPHATICS OF THE LARYNX AND NECK.

The terms "intrinsic" and "extrinsic" to indicate the anatomical site of a lesion, have long been used as a criterion as to whether conservative or radical surgical therapy should be performed in the treatment of cancer of the larynx. Reappraisal of the situation, however, carried out in the interest of preserving function without the additional risk of loss of life, has given us a new outlook. This has gradually come about as a result of studies concerned with the detailed anatomy of the organ in question correlated with observations related to the natural history of the disease. As a result, the concept of surgery of the larynx has, in recent years, undergone radical revision, and it is no longer acceptable to perform total laryngectomy for lesions, simply because they occur in surgical areas arbitrarily considered as, for example, "extrinsic."

Cancer of the larynx spreads, as do all other cancers, by one of three routes: direct extension, invasion of the vascular system and via the lymphatics. We are, in this discussion, primarily concerned with the lymphatic spread. Hajek[1] described fundamentals of laryngeal anatomy having great bear-

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†From the Department of Surgery, Division of Head and Neck Surgery, University of California School of Medicine, Los Angeles, Calif.

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ing upon the subject at hand. He pointed out that pathways of the lymphatics of the larynx follow definite patterns which correspond to our own observations pertaining to the submucosal compartments of the larynx.[2] As a result, the general concept emerges that there is ample anatomical justification for performing less than total laryngectomy in a great many cases in which the more extensive operation had previously been considered necessary.[3]

It has previously been pointed out that cancer of the larynx, extending into the deeper structures and involving the submucosal departments, tends to be limited, at least in its early stages, to a specific area which is ordinarily unilateral and, unless the mucosa is involved, this distribution remains limited for a long period of time. When the mucosa is involved, as the primary site, the spread can be more diffuse, involving both sides, but at the same time, the lesion is more apt to be superficial.

The lymphatics of the larynx originate in two principal plexuses; a deeper layer which is unilateral in distribution, and a superficial or mucosal layer, which spreads as a single system over the entire interior of the larynx without any anatomical barrier at the midline. This anatomical barrier results[2,3] from the development of the larynx at two sites of origin which fuse in the midline to the embryonic intrathyroid cartilage, with a corresponding fibrous raphe joining the lateral halves of the soft tissues concerned. It has not been established whether this applies solely to the limits of the thyroid cartilage or whether it includes the area of the cricoid cartilage as well. The former is more likely.

For the most part, therefore, the lymphatics of the larynx form pathways which merge on the corresponding side of the larynx to enter the general lymphatics of the neck by two principal pathways: one is a series of afferent tubules which emerge through the cricothyroid membrane; the other, a series of comparable channels which emerge through the thyrohyoid membrane. The former drains those areas related to the subglottic region of the larynx, overlying the cricoid cartilage beginning at its lowermost border and con-

tinuing upward. Below this level there is an entirely different pattern of flow corresponding to the lymphatics of the trachea which spread through the structures of the neck into the mediastinal rather than into the cervical nodes. This distinction is of most significant importance, since it is extremely unlikely that any extension of malignant lesions involving the peritracheal lymphatics can be considered as isolated to the structures of the neck and must be considered as metastasizing primarily into the mediastinal glands. On the other hand, lesions arising at or above the lowermost level of the cricoid cartilage are carried by a pattern of lymphatics which spread upward and diverging as they do through the cricothyroid membrane, enter lymph nodes in the upper portion of the neck where they are available to routine surgical attack.

Above the level of the cricothyroid membrane, the deeper lymphatics are found in plexuses which unite and converge to leave the interior of the larynx through the thyrohyoid membrane by afferent tubules. These channels are variable in number and form a plexus which enters the lymph nodes by more than one afferent tubule. It is, therefore, possible for a single lymph node to act as host to channels from below, representing the afferent collecting branches which have merged from the cricothyroid membrane, as well as branches above, which have emerged from the thyrohyoid membrane.

It is unlikely that any significance as to the site of origin within the larynx can be determined by the locale of a secondarily involved lymph node. Insofar as the spread of such a lesion is concerned, it would seem to matter little whether its locale is at the level of the vocal cord or below, just so long as it does not lie inferior to the lower border of the cricoid cartilage. This is extremely important from a practical point of view since, in many clinics, the presence of the lesion below the level of the vocal cords (which has been given the time-honored name of "subglottic") is ordinarily considered an adequate indication for total extirpation of the larynx. This anatomical level cannot reasonably be accepted as the dividing line between conservative and radical treatment, and from the standpoint of dissemination via the

lymphatics there is no reason to perform arbitrarily the radical procedure.

While opportunities are not ordinarily available to observe individual afferent lymphatic channels in the human, there is ample evidence in the experimental animal, such as the dog, to indicate that a confluence of tubules from the cricothyroid membrane enters the lower pole of the adjacent lymph nodes, and a second confluence of afferent channels emerges from the thyrohyoid membrane, enters the upper pole.[3] From the lymph node, efferent channels extend downward along the carotid sheath to the lowermost node of the cervical chain. Pathways by which these channels carry lymph from one node to the next can be several in number, usually from branching of a single efferent channel. The large principal efferent channel can by-pass the next succeeding node, or it can send small branches into it; however, we have not been able to observe, either in man or in experimental animals, the entrance of a principal afferent channel directly into a secondary node and its termination therein. The principal efferent channels join to form the cervical lymphatic trunk, which on the right, is called the right lymphatic duct. These terminate in the right subclavian vein at its junction with the right internal jugular vein. Valves at this site prevent the entrance of blood into the lymphatic channels. On the left side, the left jugular trunk ordinarily enters the thoracic duct, which opens into the angle of the junction of the left subclavian vein and the left internal jugular vein; but it is not uncommon to find this left cervical trunk opening independently into one of the two blood vessels named.

From these descriptions one would conclude that the right and left principal efferent lymphatic channels are independent of each other and surmise that metastases spreading from the larynx into either the right or the left side would remain localized at that particular side. This is by no means true. Numerous lymphatics pass transversely across the midline following the course of the blood vessels. This accounts for the observed involvement of the contralateral nodes[4] and negates, in some measure at least, the total protection pre-

sumably offered by unilateral radical neck dissection corresponding only to the anatomical side of the lesion. The likelihood of involvement of the contralateral nodes is increased when the lymphatics of the side involved are occluded. There is ample experimental evidence, as well as clinical evidence,[3,4] to indicate that metastases to the opposite side of the neck are clinical complications which must be considered in every case.

Apart from the pitfall of failure to identify spread of the lesion from one side of the neck to the other, one must not overlook the possibility of metastases having taken place in lymph nodes which are not identifiable, either by clinical examination or at a time of the surgical procedure. These lymph nodes, for the most part, are located in the lower segments of the neck, the nodes of the upper portion being more superficial and, therefore, more readily palpable. For the most part these more inferiorly situated nodes are located deep to the thyroid gland representing the "retrothyroid nodes." Others, more laterally placed, lie under the sternocleidomastoid muscle. This muscle, converging toward the midline as it becomes lower in the neck, is much more apt to cover and hide nodes in the lower portion than in the upper portion.

For these reasons, we have considered it imperative that no laryngeal lesions, save those at the margin of the vocal cords and which have obviously not extended beyond their local site, should be operated upon through any incision except one which gives complete exposure of those areas of the neck above mentioned. In every instance it is considered necessary to palpate the entire cervical chain of nodes from the angle of the mandible to the clavicle, with palpating fingers deep to the sternocleidomastoid muscle and the lobe of the thyroid gland. Only by this means can the presence of nodes in these obscure areas be identified, and any conservative operation carried out with assurance of good results. One simply cannot foretell, by clinical preoperative examination, when the regional nodes are involved. While lymphatic nodes can be involved in metastases so small as to be unrecognizable even at surgery, there needs to be some dividing line between

conservatism and radicalism insofar as removal of the lymphatic bearing area of the neck is concerned. We, as have most other investigators, accepted the palpability of a lymph node as the criterion which for practical purposes separates lymphatic involvement from non-involvement. This obviously provides a false sense of security in many cases, but until observations by a different method to determine metastatic processes is developed, deciding the issue at the time of surgery, with the neck open, and the critical areas palpated, appears the best method now available.

Of great interest, and possibly of considerable practical importance, is a recent observation made upon experimental animals related to direct communication between lymph nodes and blood vessels. These have been reported in more minute detail in a separate contribution.[5] By the introduction of air and saline successively into the lymph nodes of the neck we have, in a motion picture, been able to demonstrate that direct communication exists between the lymph nodes and the blood vessels of the immediately adjacent area. This is not surprising in view of the fact that the lymph nodes contain a rich plexus of the capillaries; however, previous demonstrations of these communications have not been made in the living animal, probably due to the difficulties involved from a technical standpoint in observing materials entering the rapidly moving blood stream, especially since the color of the blood prevents the observation of the entrance into it of such experimental material as dyes. The use of air and saline combination, however, makes bubbles which pass from the node into the blood stream, and these are easily recognizable. There is little or no question that this phenomenon occurs, and it would seem from repeated observation, that the pressure required for such materials to enter the blood stream directly is less than that required to enter the efferent channels of the corresponding lymph node. Such observations would of course go far to explain the presence of cancer cells in the blood stream, particularly in the area of the local lesion.

In the experimental observation concerned with matters pertaining to this report, it has been recognized that a change

in concept must be adopted concerning the nature of the lymph node itself. One ordinarily thinks of this as a filter, but actually, and in fact, it would seem to be more of the structure and functional nature of a sponge. Materials can be squeezed from it readily and with only slight pressure. Simply the weight of the examining finger upon a distended lymph node is sufficient to "squirt" the content of the node not only into the efferent channel but into the blood stream itself; and practical importance is laid upon this from the standpoint of avoiding, whenever possible, pressures upon nodes containing malignant cells either during clinical examination or during surgery.

In evaluating many years of clinical observations related to cancer of the larynx and correlating them with the described anatomical observations, the conclusion is inescapable that in many cases of carcinoma of the larynx, radical and total laryngectomy with or without neck dissection has been performed when something less than total extirpation is entirely adequate. Clinical observation of the results of our subtotal laryngectomies with reconstruction bears this out. On the other hand, we have come to the conclusion that many failures have resulted from depending upon superficial clinical examination of the neck to determine the presence or absence of spread into the lymphatics of the neck, and have been forced to the conclusion that this is an entirely inadequate method of deciding this important issue. This must be decided at the time of the surgery. The small vertical incision made over the thyroid cartilage for splitting that organ for the purpose of laryngofissure, does not provide adequate opportunity for proper evaluation of the status of the regional lymph nodes: proper evaluation is possible only through such incisions as the horse-shoe shaped incision which permits elevation of a large flap. This is the routine we have used for several years. Other incisions which allow equal access to the nodes deep to the thyroid gland or to the lower third of the sternocleidomastoid muscles, as well as those in the upper limits of the chain, must be considered equally adequate in this respect. We feel that almost any lesion of the larynx which does not involve the aryepiglottic fold, or

which does not extend below the level of the cricoid cartilage, regardless of whether or not the lesion is limited to one side of the larynx, is oftentimes resectable by a subtotal laryngectomy permitting reconstruction of the larynx sufficiently well to provide a normal channel for air adequate for purposes of speech even if not for respiration. In most cases, however, a respiratory channel through normal pathways can be provided as well. Paradoxical as it might seem, in cases in which a lesion is limited to one side of the larynx, even if relatively extensive, and no gross involvement of the contralateral nodes can be demonstrated, there is, in our experience, good anatomical and clinical evidence to suggest that a perfectly logical procedure is to perform a unilateral subtotal laryngectomy with unilateral radical neck dissection. The death rate would probably not be significantly higher and the lessened disability obviously highly desirable.

It can be concluded that studies based upon the anatomy of the lymphatics of the larynx and the neck provide a sound logical method of evaluating the indications for any one of the many surgical procedures recommended for the treatment of cancer of the larynx. Similarly, the observation that the deeper lymphatics of the larynx tend to be unilateral in their distribution, whereas the superficial lymphatics spread across from one side to the other, gives us a sound basis for evaluating the extent of the surgical techniques required in any given case. Of importance in such an evaluation is the fact that the flow of lymphatics from the inferior portions of the larynx beginning at the lower border of the cricoid cartilage is invariably upwards, whereas from this level down, the extension is into the lymphatics of the trachea and the thorax.

It is, therefore, logical to look upon involvement at the lower level of the cricoid cartilage rather than the subglottic level as representing the dividing line between the necessity for performing total extirpation of the larynx or a subtotal operation with attempted preservation of function.

Of vast importance in obtaining the best possible results for subtotal extirpation, is performing the operation via an

incision which gives access for palpation of the cervical nodes of the carotid sheath from the mandible to the clavicle.

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AMERICAN LARYNGOLOGICAL ASSOCIATION AWARDS.

The DeRoaldes Award of the American Laryngological Association was presented to Dr. Frederick T. Hill, Waterville, Maine, in recognition of his many contributions to the specialty of otolaryngology and his services to the medical profession.

The Newcomb Award was presented posthumously to Dr. Chevalier Lawrence Jackson in recognition of his outstanding work in the field of bronchoesophagology and for his untiring devotion to the furtherance of cordial relations with the medical fraternity of South America.

THE PROBLEM OF WEIGHTLESSNESS: OTOLARYNGOLOGICAL ASPECTS.*†

PAUL A. CAMPBELL, Colonel, USAF, MC,

Brooks Air Force Base, Texas.

With the advent of space flight, man will, for the first time, have to cope with problems produced by weightlessness. To be sure, we all have experienced weightlessness or zero "g" for periods of a fraction of a second, or possibly a few seconds, through free-fall, passing over the tops of roller coaster arcs, etc. Those of us who have flown Keplerian parabolas in aircraft have possibly experienced the conditions for several seconds to almost a minute. With the advent of manned rocket vehicles capable of ballistic trajectories above the sensible drag of the atmosphere, this time has been lengthened into minutes. With the achievement of manned orbital flight the occupant or the crews of the space vehicle will be weightless, following burnout of the rocket fuel, so long as the vehicle is in orbit or ellipse, and will remain weightless until acceleration or deceleration, either linear or tangential is applied, either through energy application, through drag produced by encountering resistance, or through collision with extraterrestrial dust particles, or other material.

An orbit is established and defined when a vehicle, above the atmosphere, reaches a speed of approximately‡ five miles per second and is properly positioned parallel to the surface of the earth. At this speed or higher speed when there is no drag, acceleration or deceleration, the centrifugal or fly-away-from-earth tendency produced as a function of speed and positioning, is exactly counterbalanced by the fall-to-earth

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†The contents of this manuscript reflect the personal views of the author and are not to be construed as a statement of official United States Air Force policy.

‡Depends on distance from earth's center of mass.

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tendency produced by the earth's gravitational pull. Since in this condition all inertial and gravitational forces add to the sum of zero, the condition is often spoken of as zero "g." In this situation the vehicle and everything on board, including the occupants, are weightless and will remain so until the situation changes.

Geotropism (orientation to gravity vectors) seems an inherent characteristic of all living cells, either plant or animal. Witness, for instance, the effort of the fallen tree or plant to exert great natural forces to reorient itself along a gravity vector, even though the cue of sun position is absent. Differential mass of different portions of cellular structure is thought by many to result in this geotropic or geotactic response. Regardless of cause, the phenomena are real and are deeply ingrained in the processes of life.

To get a little closer to the genus Homo sapiens, we, our ancestors, and their genes, have evolved in an environment of 1 "g"—the surface gravitational pull of the mass of the earth. When a body is static on the surface of our earth, the weight of the body is equal to 1 "g."

To get into the realm of the otolaryngologist we must point out three general conditions which occur during the weightless state:

1. The response to functions which are gravity-oriented is altered.

2. Fluid forms free floating spherical masses due to surface tension phenomena.

3. Gases are not subject to mixing or movement through convection or settling, as these are gravity-dependent processes.

Whether or not one believes completely in all of the aspects of the so-called balance triad hypothesis, I suppose is a matter of opinion. I believe most of us recognize it as a workable hypothesis. It has been especially workable in studies of orientation in relation to aviation. The hypothesis of the

balance triad dates back many years and, as we all remember, generalizes that there are three elements in effect:

1. The visual apparatus with its nervous connections.

2. The vestibular apparatus with its nervous connections.

3. The so-called kinesthetic apparatus consisting of receptors situated in muscles, tendons, joints, skin, viscera, etc.

According to the original hypothesis, two of the three systems had to function properly or disorientation would result. It is my belief that research in relation to aviation has demonstrated the dominance of the visual function and has taught us that so long as the eyes can fix upon the horizon or horizon-simulating instruments, there is no disorientation in the trained normal person.

Two of the elements of the balance triad—the utricular portion of the vestibular system and the receptors of the kinesthetic system—seem gravity-oriented, and orientation appears to function equally well whether the gravity vector is due to mass attraction or due to acceleration. The question, then, for those of us in the space medicine effort is: "What is the situation during the weightless state?"[1] At the USAF Aerospace Medical Center knowledge so far has been restricted by parabolic flights of only 53 seconds or less (there have now been hundreds of these), in which the visual cue was present and in which there was a seat belt restraint, although the restraint simply attached the observer to a gravity-free platform.* The answer determined in these runs of less than 53 seconds is that in observers with considerable air experience, little if any disorientation occurs. What will happen during sleep or during long periods of weightlessness amounting to hours or days is still an important question.

To simulate weightlessness, a group of pilots and trained observers are flying F-100 aircraft assigned to the School of Aviation Medicine of the USAF Aerospace Medical Center. The aircraft are instrumented for the studies. Figure 1 shows the Keplerian flight profile, the concept of which was

*The Project Mercury astronaut was weightless for a matter of minutes although exact data has not been published as yet.

introduced as a possible method of simulation of the weightless state by Fritz and Heinz Haber in 1950,[2] and discussed in greater detail by S. J. Gerathewohl, O. L. Ritter and H. D. Stallings in 1957.[3] According to the profile in Figure 1 an ideal limit of 63 seconds of weightlessness would be accomplished. This ideal has not been accomplished to date as it entails smooth accelerative change during entrance of the afterburner boost. Smooth acceleration through this phase is

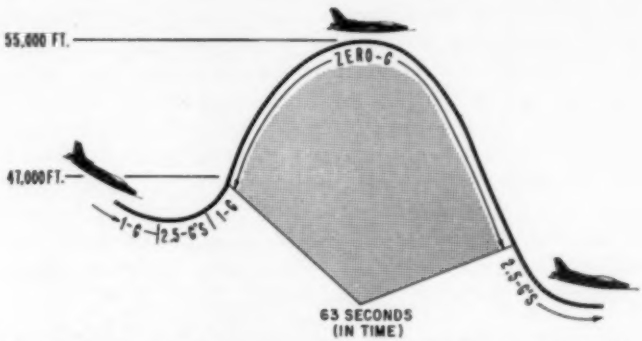

Fig. 1. Parabolic flight pattern. Diagram showing flight profile of F-100 aircraft during zero "g" maneuver. Hypothetical time of weightlessness should be about 63 seconds. In practice, only 53 seconds has been achieved.

extremely difficult. Thus, 53 seconds is about the longest period achieved.

To illustrate some of the problems of weightlessness I now present a short movie which first demonstrates the Keplerian parabola as it is flown in F-100 aircraft.* The movie then shows the problem of fluid control. Due to surface tension phenomena the particles of fluid are drawn together into least volume and thus assume spherical formation. The globules of fluid in their ameboid movements float about the cabin much as any other free body. During inspiration the material can get into the nasal sinus cavities through venturi effect. The solution to the fluid-taking problem is then demonstrated

*For this movie I am indebted to my colleagues, Major Royce Hawkins and Captain Frank Young.

to be the squeeze bottle, which is used to inject the fluid into the posterior pharynx, where it can amply be cared for by the processes of deglutition.[4]

In further analysis of the movie, it is obvious that there is no disorientation of the occupants of the aircraft. They are restrained by their seat belts and have visual cues. The gold fish shown in the container present an interesting study as they are not restrained. They do have visual function, but this is probably seriously interrupted by the movements of the aircraft and the difference in the indices of refraction of the various media surrounding them. One must be careful in interpretation of simple experiments of this type; however, the reactions of the fish are interesting. First, some excitement seems evidenced during the periods of positive acceleration at either end of the parabola. During weightlessness they are not excited and, above all, do not panic. They undergo slow swimming movements, rolling backward and forward, or spiraling upward or downward. I believe the best description of these movements is that of the "hunting effect"—hunting for the gravitational vector.

The use of forces of acceleration[5] through rotation of the space vehicle has been discussed by Gail, Oberth, Noordung, Tsiolkovsky, von Braun, and others, for gravity simulation or substitute. Von Braun,[6] several years ago, for instance, discussed a space station to be assembled in orbit in the form of a huge doughnut some 200 feet in diameter, which could be slowly rotated and thus would produce sufficient "synthetic gravity" to furnish minimal cues and to serve other mechanical requirements. It is, however, not difficult to imagine the inherent technical difficulties.

Now a word about stagnation of respired gases. Mixing of air or respired gases by convection in the usual fashion is gravity dependent because some gases are heavier than others; furthermore, in a given gas in the presence of gravity the warmer portions of the gas rise while the heavier portions fall. The question then is: "What is the effect of this stagnation on the constitution and distribution of gases being respired?" The problem, however, is not a serious one, as

the solution is quite simple. By means of even simple apparatus, cabin air and gases can be kept moving and thus mixing; consequently obviating the problem.

In conclusion, the otolaryngological problems resulting from weightlessness during space flight are not serious, with the possible exception of the problems of orientation during periods when restraints are not in place or during periods when visual cues are absent. The use of acceleration produced by turning the space vehicle about an extended axis has been suggested by several. Technically, such simulation is difficult, but with advances in the state of the art, may be possible.

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THE BRAIN-INJURED CHILD WITH IMPAIRED HEARING.*

HOLLIE E. MCHUGH, M.D., C.M.,†

Montreal, Canada.

The problems that relate to hearing and language disorders in children are many and varied. Clinicopathologic studies that correlate pathology in the inner ear and in the brain with well documented clinical examinations are limited, or practically non-existent. We are tempted, therefore, to seek a deeper understanding of the brain-injured child with impaired hearing, in a review of the data obtained from the numerous disciplines that have a common interest in the effects of brain damage and sensory deprivation. Continuing research in neurophysiology, neurosurgery, electroencephalography, clinical and experimental psychology, audiology and otology, has provided information that may be applied, with considerable benefit, to our concept of the problems that relate to these particular children. Any brief account of such an enormous topic must, of necessity, run the recognized risk of oversimplification.

THE TERM—BRAIN-INJURED.

Although the term "brain-injured" is vague and unscientific, it is used herein for want of a better title for those children who cannot, as yet, be conveniently diagnosed as any specific type of brain "damage". The isolated or sometimes irregular difficulties that they exhibit in the development of verbal or other forms of language, nevertheless, appear to be the effects or subtle expressions of minimal brain damage or dysfunction. Children with cerebral palsy and mental retardation are, therefore, not included.

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†From the Hearing and Language Disorders Study Group, The Montreal Children's Hospital, and McGill University, Montreal, Canada.

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These children are a serious problem to their parents and all too often to the many people who attempt to make a specific diagnosis of their problems. If the parents hold high expectations for their offspring, or are rigid and demanding, the tensions, anxieties, frustrations, and sometimes hostility so engendered may compound the child's difficulties to the point where an entirely new set of symptoms appear, to confuse the diagnosis further. These parents need to have their feelings of guilt allayed, and they need support. The parents also need mature guidance and assistance in acquiring an understanding of the emotional and educational problems involved, and in this respect the otologist may play an important role.

THE BRAIN-INJURED CHILD.

In the study of brain function, it is apparent to all that despite the differentiation there is a certain unity of activity that is essential for the development of the uniquely human power of using symbols—the power of speech that makes man "lord of the earth."[11] In the study of these children one is impressed by the multiplicity of the effects of brain injury, from whatever cause, in terms of the disorganization or disunity of function and the unpredictable responses to sensory stimuli, be they auditory, visual, tactile, kinesthetic, or even pain. The variety of clinical manifestations depends of course upon the site, severity, and the time of the brain damage.

In this context the brain-injured child is one whose brain was "damaged" as the result of anoxia, trauma, toxins or infection, before, during, or at any time after birth.[22] The "damage" may have been minimal or severe, focal or diffuse, cortical or subcortical, and/or in the brain stem. There is now important experimental evidence[21] to support the clinical suspicion that minimal lesions in the midbrain may be of major significance in these children.

It is somewhat paradoxical to note that the progress in medical treatment that has permitted the survival of many children from previously fatal accidents and diseases has, at the same time, resulted in many having permanent handicaps.

There are many who have acquired severe hearing losses or total deafness following the administration of antibiotics known to be ototoxic, that presumably could have been prevented.

These brain-injured children present a characteristic pattern of behavior.[16] In general, they are inconsistent, unpredictable, and emotionally unstable. They are distractible; on the other hand they may persist in some activity long after it would seem to have any meaning, *e.g.*, they tend to perseverate. Their hyperactivity seems to lack the purposeful activity of a deaf child. Mild motor incoordination and autonomic disturbances—irregular bladder and bowel control, excessive salivation and perspiration—are not uncommon. These behavioral abnormalities may be minimal, difficult to detect, or very conspicuous.

These children may have variable degrees of difficulty in the interpretation and use of signs and symbols. The normal trend of infantile vocal activity to verbal activity in the emergence of language is delayed. "The real problem of speech, however, is not vocalization, but the capacity of manipulating ideas or symbolic acts."[5] These children do not easily acquire the meaning or significance of words whether spoken, written or read. They may be said to have a language disorder: a disorder in symbolic behavior. Those with more sharply defined difficulties have been called aphasic or aphasoid, depending upon the severity of the symptoms exhibited.

True aphasia is a relatively rare condition in children. The term congenital aphasia has been used by some, without apology,[10] because the behavior of a child who has lost normally acquired speech and its understanding, is often indistinguishable from one with retarded language development.

HEARING AND ATTENTION.

These children differ in other respects. Some are able to hear but they are unable to listen, because of a disturbance in the complex mechanism of attention. They cannot respond to what they hear (their sound world) selectively and mean-

ingfully. They appear to respond to all sounds as though they were of equal importance simultaneously; as a result they are easily distracted and unable to maintain attention. The constant bombardment by auditory stimuli appears to be a threat to the adjustment and survival of this type child, and he may react by apparently not listening. His responses to sound become inconsistent, unpredictable, and he may appear to be deaf.[16]

In recent years there has been a gradual convergence between neurophysiological and psychological evidence in this general area of interest, and it is now possible to correlate certain electrophysiological phenomena with behavioral changes. Lashley[12] stated that the problem of attention is the problem of the selective dominance of some group of related neural activities with the simultaneous suppression of others. The "focusing of attention" requires a selective facilitation of certain sensory input above other sensory afferents.[23] In the neural elements that normally respond to auditory stimuli, the efficiency of response is apparently increased with "attention." The effect is complex, but there appears to be an increase in the signal to noise relationship, *i.e.*, a change in relation to the reception of specific auditory stimuli. In the study of cortical unit activity in cats, Hubel *et al.*,[6] have discovered a "population of cells that appear to be sensitive to auditory stimuli only if the cat 'pays attention' to the sound source." The question of just where attention units occur in and near the auditory cortex remains to be settled. The human brain is constantly acted upon by a variety of stimuli through the sense organs, but only a small proportion of these elicit observable responses. Although attention is an elusive variable that no one has as yet been able to quantify, there appears to be an optimum degree of electrical activity in the auditory system that is necessary for maintenance of normal efficiency. When the system is over-driven there is either suppression of the stimuli or over-excitation with generalized disturbances in behavior. The reticular system and certain parts of the thalamus seem to play an important role in the mechanism of attention.

The ability to pay attention demands, therefore, a certain

organic and functional integrity of the central nervous system that appears to be lacking in many of these "brain-injured" children. It is important to recognize that their lack of attention may be a symptom or an expression of a disorder, and not necessarily a voluntary or willful act.

IMPAIRED HEARING.

Impaired hearing herein refers to a partial loss of the sense of hearing, not a profound loss or total deafness. In these particular children abrupt high tone losses, or moderate flat sensory neural losses, seem to have a more disturbing over-all effect on their ability to learn verbal language than the same degree of partial sensory deprivation would have in a child with an undamaged brain. It is difficult to discover, at this stage of our knowledge, whether the flat 30 to 40 decibel losses that are not uncommonly seen, are due to a peripheral neural lesion or to some as yet unrecognized cause in the auditory pathways. When this degree of presumably peripheral hearing loss is increased by a conductive component of 20 to 30 decibels, due to middle ear inflammation, the result may be disastrous in terms of the child's general and communicative behavior, unless promptly recognized and treated.

THE EFFECTS OF SENSORY DEPRIVATION.

Our knowledge of how a sound stimulus is analyzed, transmitted, perceived, remembered, or integrated and synthesized into speech, thought production, or emotional response and appropriate motor activity, is still very incomplete.

It is certain, however, that damage to this complex auditory system in children is not to be evaluated or expressed in terms of a decibel loss: there must be an interpretation of the total effect upon the child's sensory, perceptual, intellectual, emotional and motor behavior in order to understand his problems.

Experimental psychology and space medical research have shown the very great disrupting effects of sensory isolation.

It has been demonstrated that the lack of sound and other sensory stimulation has a profound effect on human behavior,[5] and the modern surgical techniques that permit the dramatic restoration of severe conductive deafness to normal hearing levels, have also been found to produce far greater effects on over-all brain activity, than were ever previously suspected.

Much interest has centered, in recent years, around brain stem structures and their potential role in behavior. Particularly important in current thinking is the fundamental work of Magoun[13] and his colleagues on the central core of the brain stem, the multineuronal, reticular formation, which receives afferent supply from all sensory systems and contributes significantly to the activation of the cortex and other forebrain structures. This system, including the nonspecific thalamus, has also been implicated in the neural mechanisms for emotion, motivation, learning, and perception and even in consciousness and the integration of the highest functions of man.[20]

Sprague, Chambers and Stellar,[21] in experiments on cats, undertook to make a direct attack on the problem of the respective roles of the reticular formation and the sensory pathways in behavior. Their studies suggest that "it is the specific, the patterned and localized, sensory information carried to the forebrain via the lemnisci, that is essential." They report that lesions of the lateral portion of the upper midbrain, involving medial, lateral, spinal, and trigeminal lemnisci primarily, result in a consistent syndrome of symptoms in the cat. *1.* There is a marked *sensory deficit*, characterized mainly by sensory inattention and poor localization in the tactile, proprioceptive, auditory, gustatory, and nociceptive modalities, where direct pathways are interrupted. Similar defects appear in vision and olfaction where no known paths are interrupted. *2.* There is a *lack of affect*, with little or no defensive and aggressive reaction to noxious and aversive situations and no response to pleasurable stimulation. The animals are mute, lack facial expression, and show minimal autonomic responses. *3.* They show a *hyperexploratory activity* that appears to be centrally directed and is very diffi-

cult to interrupt with environmental stimuli. 4. They also demonstrate *exaggerated oral activities*. In interpreting these results the authors emphasize the view that the syndrome is due chiefly to the extensive, specific, sensory deprivation produced by interruption of the lemnisci at the rostral midbrain, "a deafferentation of the forebrain which greatly impairs normal neocortical function."[21]

Fig. 1. The various disciplines involved in the Hearing and Language Disorders Study at the Montreal Children's Hospital (from the movie, The Evaluation of Hearing in Pre-school Children Who Lack Normal Speech, by H. E. McHugh, M.D., and R. H. McCoy, M.D.).

In addition to other important sources of evidence, the behavior of their cats with lemniscal lesions suggests the importance of rich and varied sensory stimulation, in the development and maintenance of attentive, affective, and adaptive behavior. While the parallelism is purely speculative, the resemblance of the behavior of the cats with midbrain lesions to the behavior of certain autistic children is remarkable. The very recent report of this work is a monumental contribution to our understanding of sensory neural mechanisms and of the importance of the midbrain in the integration of sensory stimulation and behavior.

A QUALITATIVE METHOD OF EVALUATION.

In 1950 a project was initiated by the author at the Montreal Children's Hospital,[14] the stated purpose of which was to "establish, maintain, and expand a clinical unit for the study of hearing and language disorders in children" (see Fig. 1).

This program was planned as a means of overcoming the isolation of the various specialties involved in the diagnosis, treatment, and education of these particular children. An effort was thus made to integrate the numerous medical and associated disciplines with this common interest, and it was hoped that such a study would create an atmosphere favorable to learning and to an exchange of knowledge that would be a benefit to the children with these serious handicaps. As this study has continued, inevitable changes in concepts have occurred.

This method of investigation has proven to be a constructive step toward an understanding and practical means of handling the complex problems that relate to these children.[15]

THE LACK OF NORMAL SPEECH DEVELOPMENT.

A preliminary report on this study revealed that 42 per cent of the 60 children investigated had multiple handicaps.[14] Diagnosed on the basis of what was considered to be the major handicap, the lack of normal speech development was due to peripheral deafness in 50 per cent, brain injury—29 per cent, mental retardation—13 per cent, and severe emotional disturbances (infantile autism and schizophrenia)—8 per cent (see Fig. 2).

The 450 children studied since that time may be divided into the same categories, in approximately the same percentages, the only exception being an apparent increase in the number with evidence of minimal brain damage, combined not infrequently with a significant peripheral hearing loss.

Before discussing a few selected cases from this study, it

would not be amiss to review very briefly some essential details of the auditory system.

THE SENSE OF HEARING.

The human ear is a remarkably sensitive and efficient sense organ, even when it is compared with the most elaborate electronic recording instruments. It collects, transforms, and

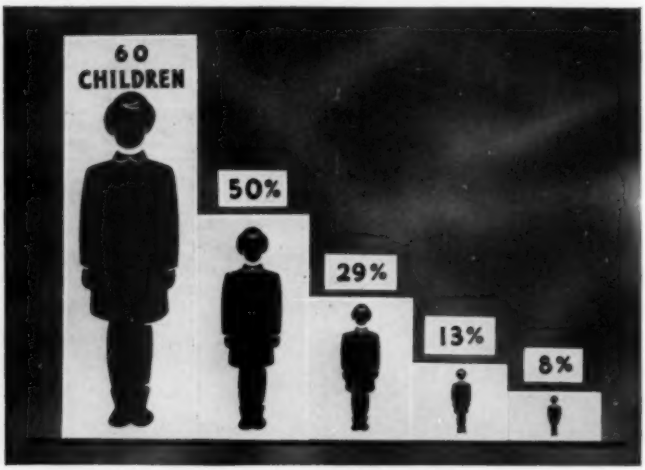

Fig. 2. The percentage incidence of deafness, brain injury, mental retardation, and severe emotional disturbances, in children who lack normal speech development (from the movie mentioned in Fig. 1).

analyzes complex sound waves with a range of frequencies from 16 to at least 20,000 cycles per second. The vibrations of the stimulating sound source are increased in intensity and decreased in amplitude by the transformer mechanism in the air-filled middle ear space: the vibrations are thus transmitted to the fluid filled scalae of the inner ear, where the travelling waves produced along the cochlear partition selectively stimulate the hair cells of the organ of Corti. The energy of the original sound wave complex is thus converted into nerve impulses. Our knowledge of how vibrations or

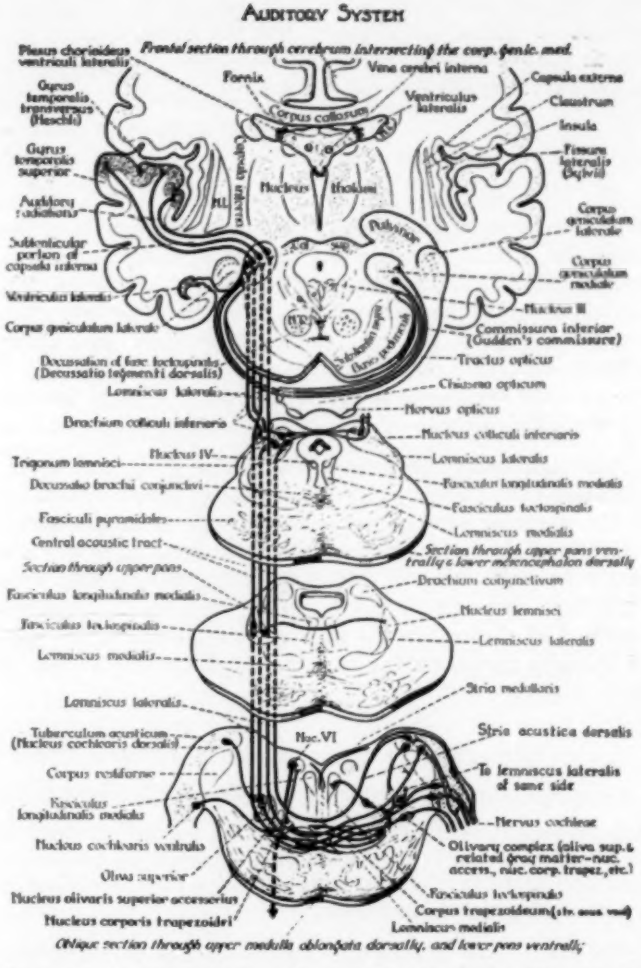

Fig. 3. The Auditory System (from A. T. Rasmussen, The Principal Nervous Pathways, with permission of the publishers, The Macmillan Company, New York).

mechanical motions in the cochlea trigger electrical potentials has been enhanced, in recent years, by the extraordinary studies of Georg von Bekesy,[1] but the exact mechanism by which the incredibly delicate sensory hair cells produce electrical nerve impulses is not yet known.

THE AUDITORY NERVOUS SYSTEM.

The nerve impulses that arise in the sensory hair cells pass through the processes of the bi-polar ganglion cells of Corti, along the VIIIth nerve to the groups of dorsal and ventral cochlear nuclei, in the medulla oblongata[19] (see Fig. 3). These nuclei are subdivided into smaller complexes having characteristic cell-types, afferent collateral endings, and efferent connections. The dorsal cochlear nucleus is laminated like the cerebral cortex, while the ventral is not.[3] Only the barest morphological details of this complicated area are known.

Although some nerve impulses ascend from this level on the same side, the majority cross over in the trapezoid body to the superior olivary nucleus, and ascend in the afferent tracts to the auditory cortex, with relay stations in the nuclei of the lateral lemniscus, inferior colliculus (where a second important decussation takes place) and medial geniculate bodies, and thence via the auditory radiations to the cortex in Heschl's gyrus, on the supero-medial surface of the temporal lobe of the brain.[19]

THE AFFERENT AND EFFERENT PATHWAYS.

Continuing studies of the very important reticular formation indicate that alternate afferent and efferent, nonspecific, routes exist as shown schematically in Figure 4. An auditory pathway to the cerebellar vermis has also been described, the function of which is unknown.[4]

The right side of this drawing shows the very important descending, or efferent, system of neurons. These apparently originate in the cortex, descend with relays in each of the nuclei of the ascending tracts, to terminate via the efferent olivo-cochlear bundle (of Rasmussen) in or near the internal

hair cells in the cochlea. Galambos has shown, in animal experiments, that stimulation of this bundle inhibits or prevents impulses passing from the cochlea to the cortex. This suppressor system may play a role in the neural mechanism of listening. The nervous mechanism of figure-ground organization, or foreground-background differentiation, is as yet unknown.[12]

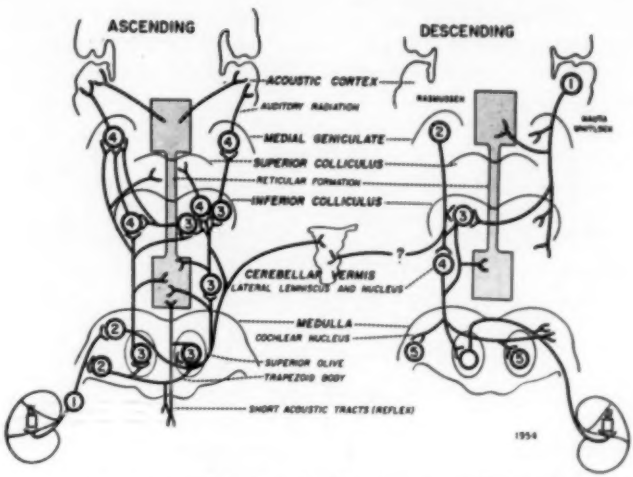

Fig. 4. Anatomical plan of auditory neurons (Galambos[4]).

THE SENSORY CORTEX AND THE CENTRENCEPHALIC SYSTEM.

In considering the auditory cortex and the localized areas that are known to be important "centers" for the receptive and expressive phases of speech, Brodal cautioned that the cortex appears to function as a whole and not alone. There is always some sort of inter-relation with lower structures, primarily the thalamus and hypothalamus.[2] The function of the cortex is, therefore, not only one of localization and integration but also a combination of both.

More recently[18] it has been pointed out that the human

cortex is made up of numerous functional areas, each of which has its most important connections with areas of gray matter in the brain stem including the thalamus. The subcortical areas of gray matter, by means of their projection fibres, serve to coordinate and to utilize the functional activities of cortical areas and to integrate that activity with the rest of the brain. Transcortical association tracts are of importance, no doubt, but certainly of less importance than subcortical integration (see Fig. 5).

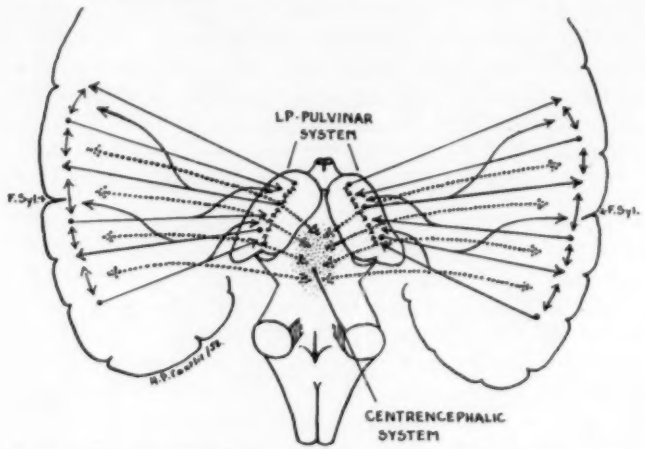

Fig. 5. Diagrammatic representation of hypothetical thalamocortical relationships of temporoparietal cortex with the LP-Pulvinar system of the thalamus, and projections to and from a centrencephalic system of the brain stem (from Epilepsy and the Functional Anatomy of the Human Brain, by Wilder Penfield and Herbert Jasper, with the permission of the publishers, Little, Brown and Company, Boston).

The visual and auditory receiving areas are, according to Penfield and Jasper,[17] the most highly specialized of all cortical sensory receiving areas in man. The primary visual and auditory sensory areas on the cortex of each hemisphere are considered by Penfield and Roberts,[18] to be transmitting areas—a way station in the stream of visual and auditory impulses.

It is important to note, at this point, that the auditory nervous system "expands" from the periphery to the cortex.

It is generally accepted that there are 30,000 fibres in the auditory nerve, about 90,000 cells in the cochlear nucleus, and more than 10,000,000 cells in the auditory cortex.[20]

The precise study of epilepsy and epileptic patients convinced Penfield and Jasper[17] that there is an area in the central nervous system devoted to the integration of the functions of the sensory and motor areas of the cortex, an area in which is to be found the "neural substratum of consciousness." The sensory pathways lead nerve impulses from the periphery to the diencephalon. In this higher brain stem there is a ganglionic interruption, but the current passes onward to specialized sensory areas of the cortex. In the cortex there is another ganglionic interruption before the current passes back to the centrencephalic integrating system, situated largely in the diencephalon and adjacent portions of the brain stem. The centrencephalic system may be defined as "that neuronal system that has a symmetrical functional relationship with the cortex of the two cerebral hemispheres," and also the "integration of varied specific functions from different parts of one hemisphere"[18] (see Fig. 5).

THE NEURAL MECHANISM OF SPEECH.

On precise evidence from cortical mapping and cortical excision, Penfield and Roberts[18] propose: the comprehension of speech occurs after receiving auditory impulses in both hemispheres and in the higher brain stem, and during the interaction of impulses between the higher brain stem and the left temporo-parieto-occipital region. Reading occurs after receiving visual impulses in both hemispheres and in the higher brain stem, and during the interaction of impulses between the higher brain stem and the left temporo-parieto-occipital region.

Impulses produced after interaction between the higher brain stem and the left hemisphere may be transferred to the motor cortex of either hemisphere, and then to the final common pathway to the muscles used in speaking; and motor speech accompanies these transactions. Interaction may occur

between Broca's area and the higher brain stem before transfer of impulses to either motor cortex (see Fig. 6).

DAMAGE TO THE CORTEX.

If Broca's area is destroyed, then interaction may be between some other part of the left hemisphere (*e.g.*, the supplementary motor area) and the higher brain stem before transfer of impulses to either motor cortex.[18]

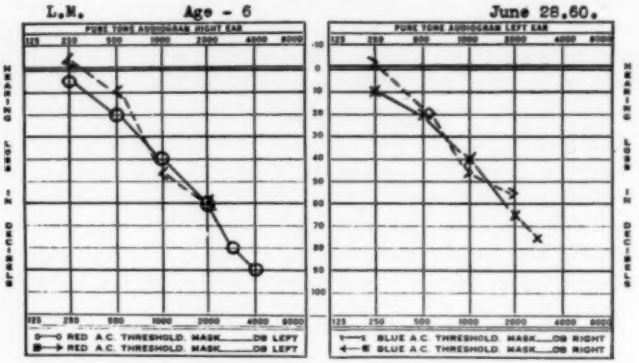

Fig. 6. Pure-tone audiogram of L.M. (see Case 1).

If the auditory area of one hemisphere is destroyed then that of the other is used alone. Transient dysphasia would result only if the lesion were on the left half, and the cells or pathways connected with the higher brain stem and used during the comprehension of speech were affected. Persistent dysphasia would occur if the latter were functioning abnormally or if the lesion were very extensive. The right hemisphere could function for the comprehension and execution of speech only after training.[18]

In the first clinicopathologic study of a well documented case of congenital aphasia[19] to be reported, Landau, *et al.*, stated "our data are sufficient to show severe damage to the primary auditory projection pathway bilaterally, with cor-

responding retrograde degeneration of the medial geniculate nuclei." They were unable to say whether a possible small amount of residual functional tissue in this system could have accounted for the subject's demonstrated capacity to develop speech and language. The cerebral lesions in this case were not limited to the auditory system, and the clinical picture could have been partially related to destruction of other structures in the sylvian region. They commented that "it would be gratuitous to infer that gross lesions of the geniculate-cortical system are likely to be found in most other cases of congenital aphasia, for a large portion of the congenital aphasic population shows no collateral clinical or laboratory evidence of such extensive lesions." They finally emphasized that "residual function is due to residual tissue; the lesion may be less constant than the adaptive mechanism that remains unchanged."[10]

Animal experimentation has shown that limited cortical injury can show widespread effects in behavior presumably controlled by cortical tissue far removed from the site of injury; and a specific behavior pattern which deteriorates after ablation of its cortical center will show recovery with the passage of time or with special training. The mechanism behind these phenomena has never been made clear. One possible avenue for these generalized effects may be biochemical.[9]

With brain-injured children it is immediately apparent that an early, exact, diagnosis is difficult, and in many cases it may be impossible until the child has reached a stage of maturity, or reorganization of this central nervous system which permits a reasonable assessment of his special communicative difficulties.

CLINICAL ELECTROENCEPHALOGRAPHIC STUDIES.

Jasper and Van Buren[7] in their clinical electroencephalographic studies, on the interrelationship between the cortex and the subcortical structures, have noted that when the cortex is deprived of afferent supply it may show a marked reduction or absence of electrical activity, or it may give

rise to abnormal discharges. They emphasized that the abnormal discharges may be due either to the intrinsic effects of denervation or ischemia upon the functional properties of the cortex itself; or the abnormal waves may be the result of the simple lack of the constant flux of afferent impulses which serve to provide both specific and unspecific activating or regulating (inhibiting) influences, and timing (synchronizing or desynchronizing), of the constant play of activity over cortical networks.

There is convincing clinical and experimental evidence that the spontaneous cortical rhythms which make the greater part of the EEG record are most sensitive to lesions affecting principally the unspecific ascending reticular system in the core of the brain stem (midbrain through the thalamus). Relatively minor effects result from lesions confined to specific projection systems, or from lesions of the brain stem caudal to the midbrain.[7]

It is well known that the alpha rhythm in the EEG tends to be arrested by visual attention or stimulation.[7] Auditory stimuli on the contrary do not alter the alpha rhythm. Experiments in progress, however, suggest that a combination of visual and auditory stimuli produce changes in the EEG record that may prove to be of clinical value in the investigation of the brain-injured child.

An EEG has been obtained on every child in this study of hearing and language disorders. An analysis of the results will be reported at a later date; in general, however, the incidence of diffuse dysrhythmia, potential epileptogenic foci, and other focal and diffuse abnormalities is much higher than one might expect with grossly normal neurological examinations.

In the brain-injured child with impaired hearing it is, therefore, difficult to know whether the observed behavior is an appropriate adaptation to the unpredictable inhibition or excitation of peripheral sensory stimuli from central structures, or from cortical structures, and hence *normal for a certain type of pathology* or *whether the behavior is abnormal, or disorderly.*

ILLUSTRATIVE CASES.

*Case 1.** L. M. Male. Age: six years, three months. Oct. 19, 1960. Referred because of delayed speech and suspected hearing loss.

History: Born: July 19, 1954. Full term. Weight: ten pounds, five ounces. Fourth child. Mother confined to bed for first three months because of persistent vomiting, anxiety, and depression. Severe pains for ten days before induced labor. Shoulder presentation. No postnatal cyanosis or jaundice. Mother stopped nursing at one month because of nervousness and depression. No feeding problems. No convulsions.

Genetic Development: Sat alone at eight months. Walked at 15 months. First words at 16 months. Short sentences at three years. Toilet trained at three years.

General Behavior: Right handed. Not awkward or uncoordinated. Appetite good. Sleeps well, but insists on someone else being in the room. Prefers to play with younger children. Distractible and hyperactive. Inconsistent in day-to-day behavior. Now makes his wants known verbally. Not sensitive to vibrations. Response to sound inconsistent.

Childhood Diseases: Chicken pox at three years. German measles at four years. Scarlet fever at five years. Three months later impaired hearing was first suspected. He was subject to frequent colds and sore throats. Occasional earaches.

Pediatric Examination: No physical evidence of disease, and particularly no CNS abnormalities.

ENT Examination: Ear drums normal. Tonsils and adenoid moderately large and mildly infected. No cervical adenopathy.

Hearing Tests: Because of the lack of attention and distractibility, five tests were performed before consistent results were obtained. The audiogram shows a bilateral abrupt high tone nerve sensory hearing loss (see Fig. 6). Speech reception threshold was approximately 40 db in both ears. Discrimination was 48 per cent in the right ear and 52 per cent in the left. Results difficult to estimate because of limited vocabulary and poor articulation.

Speech Therapy: Articulation problem in keeping with the hearing loss. When this child first attended the speech clinic nine months ago he was unpredictable in his behavior, and he had a very short attention span. He appeared to be antagonistic to the speech lessons as he preferred the Nursery School. Work on his speech during that period was primarily language development.

Psychometric Evaluation: L. was quite cooperative and showed interest in all the test items. He seemed to understand all the spoken instructions, and he performed all the tasks presented. He talked a little, but the examiner had difficulty in understanding everything he said. On nine subtests of the Nebraska, using the norms for children with hearing, he achieved a Learning Age of seven years, I.Q. 113, indicating functioning in the Bright Normal Range of Intelligence. The Performance Scale of the WISC elicited a Performance I.Q. of 104, indicating average range, as far as performance skills are concerned. The Rutgers elicited a Drawing Age of six years, five months, which is consistent with the above. His reproduction of the Bender Figures shows dissociation, substitution, overlap and errors suggesting a visual motor impairment.

*This is a brief outline of this child's record. The author has attempted to avoid any misconceptions in summarizing the various reports, and it is hoped that no injustice has occurred.

Opinion: Whether this child should continue in a regular class or attend a class for the hearing handicapped will depend on several factors, *i.e.*, hearing tests, speech progress, relationship to other children, etc. In a one-to-one relationship, as in the testing situation, he appeared to hear well enough to follow instructions.

EEG Report: There is a minimal, diffuse, slow irregularity. In addition, the response to hyperventilation suggests a subcortical instability. The changes are minimal.

Psychiatric Consultation: At age five this child was a behavior problem with temper tantrums, attacking siblings and mother with knives, stealing money from mother's purse to buy matches, etc. Both parents are married for the second time. Their first spouses were killed in concentration camps. The parents speak Hebrew in the family. This child was four before he started speaking English. He is very distractible. He seems to enjoy getting the mother upset by his aggressive behavior. His acting out behavior seems to be worse since he had scarlet fever. He still demands to have the TV louder than the others in the room wish. He was started in Kindergarten but he refused to go after two weeks. There is a definite separation problem. He was referred for screening re-admission to the special Psychiatric Day Treatment Center.

Conference—October 19, 1960.

This child has a moderately severe bilateral nerve sensory hearing loss, which undoubtedly is the cause for at least part of his discrimination, speech and language difficulties. The prenatal and birth history, the delayed speech development, the EEG and psychometric findings, and his early behavior, are all suggestive of minimal brain injury; his present difficulties are, therefore, the result of a combination of all these factors. The two languages and the persistently high level of anxiety in the home are obviously additional factors.

An assessment of this child's speech and educational problems in terms of the audiogram alone would be inadequate. He has been placed in a special class for the hard of hearing, where he will receive speech therapy, and consideration will be given to his visual motor difficulties. The mother and child will be seen from time to time by the senior medical social worker for support and guidance, and, if necessary, re-evaluation by the psychiatrist can be arranged. This whole problem will be reviewed in one year, or before if necessary.

Case 2. E. P. Age: 6 years, 11 months. Conference: October 9, 1957. Referred because of abnormal speech and suspected hearing loss.

History: Born: November 23, 1950, at 8½ months. Mother Rh negative. Labor induced. Pains for two weeks, "hard" for four days. Forceps delivery. Second pregnancy. Birth weight—6 pounds, 5 ounces. Jaundiced

for three weeks after delivery. Child received complete transfusion in first day of life. In incubator for one week.

Genetic Development: Sat alone at 6-7 months. Walked at 13 months. Slow to follow objects with eyes, 4-5 months. First words at two years, sentences at four years. Toilet trained at two years.

General Behavior: Right handed. Walks unsteadily. Appetite good. Sleeps well. Low frustration tolerance. Has frequent temper tantrums. Hyperactive. Very inconsistent in responses to sound. Voice very loud and high pitched at times.

Childhood Diseases: Frequent colds and ear infections after seven months of age. Impaired hearing first suspected after measles at two and one-half years. Hernia operation at three years. T & A at four years.

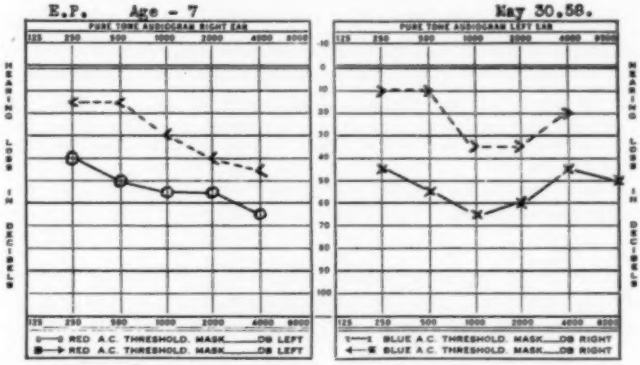

Fig. 7. Pure-tone audiogram of E.P. (see Case 2).

Pediatric Examination: No definite evidence of neurological deficit. Coordination fair, cannot tie shoelaces. Speech fairly clear. Some misunderstanding of similar words.

ENT Examination: Some thickening of both ear drums. Mucopurulent discharge in both nostrils. Sinus X-rays show some cloudiness in both ethmoids and in both antra. Tonsils removed. Nasopharynx—scattered lymphoid nodules covered with purulent discharge. Slight enlargement of cervical glands.

Hearing Tests: Three tests obtained between June 1957 and March 1960, showed a fairly consistent bilateral nerve sensory hearing loss, with a variable conductive loss. On two occasions the AC average for speech frequencies was 55-60 db in the two ears. The best BC average for these frequencies was 30 db. In March 1960 the conductive component was less (see Fig. 7). The Speech Reception Threshold was 50 db in the right, and 40 db in the left ear. Discrimination was 88 per cent right, and 80 per cent left. The discrimination scores appeared to decrease with increased intensity. "Auditory training and speech therapy both seem to be needed, whether or not a hearing aid is finally obtained."

Speech Therapy: Comprehension inconsistent. Voice slightly flat.

Speech mechanism normal. R and L blends, SH-CH, Z and S blends inconsistent. Some slurring on polysyllables. He is to continue with speech therapy. Amplification is to be tried.

Psychometric Evaluation: This child was tested in 1951. He obtained an I.Q. of 100 on the Cattel Infant Intelligence Scale. He is in Grade 1, and he is not getting along very well. He has considerable difficulty with reading and printing and tends to reverse his letters. According to the Nebraska Test of Learning Aptitude (scored according to the norms for Young Deaf Children) E. is functioning at a Bright Average Intellectual level, with a learning age of eight and a corresponding Learning Quotient of 114. His lowest score was L with Block patterns on which he scored at the 5-6 year level. His highest score was on Memory for colored objects which scored around the 10 year, 6 month level. His scores for Bead Stringing, Block Patterns, Completion of Drawings and Puzzle Blocks, items involving visual-motor coordination were the lowest, and this might indicate visual-motor problems. The Goodenough Drawing Test and the Bender Gestalt also indicate visual-motor difficulties. This undoubtedly accounts for his trouble with reading and printing. He would probably benefit more from a special educational program.

EEG Report: The EEG shows minimal evidence of focal cortical dysfunction in the left temporal region. In addition to this there is instability on hyperventilation which is not a definitely abnormal finding in a child of this age.

Conference—October 9, 1957.

This child has speech, language, hearing and educational difficulties that are undoubtedly due to the combination of a number of factors. There is a basic nerve sensory hearing loss of approximately 30 db, which is increased at times by a conductive component of 25-30 db, when there are inflammatory changes in the middle ears; his hearing, therefore, is inconsistent. The birth history, the delayed speech development, the EEG and psychometric findings, and the specific educational difficulties, all are suggestive of the compounding effects of minimal brain injury. He is to have an allergy investigation and appropriate treatment of the repeated respiratory infections which intermittently increase his hearing loss. The superimposed conductive component due to the inflammation in the middle ears converts this child from one with a borderline communication disorder, into one who requires special educational classes, unless this conductive factor can be eliminated. He will also need remedial reading assistance. He is to continue with speech therapy and a hearing aid will be tried. Further recommendations will depend upon his progress.

COMMENTS.

These two cases exemplify the complex problems involved in many of the children with speech, hearing, and educational difficulties. The advantages of a complete investigation are apparent.

It is uncertain whether there was a hearing loss before Case 1 had scarlet fever and Case 2 had measles, but the impaired hearing was first suspected *after* these infections, and in both cases the illness was severe. There were no convulsions in either case. The additional sensory deprivation certainly changed the behavior of Case 1. One might speculate, on the basis of experimental evidence, that this degree of increased sensory deprivation has a far greater effect on the behavior of the child than one might otherwise suspect. When compared to other children, with no evidence of brain injury, but with the same degree of hearing loss, there is a remarkable difference in their ability to communicate verbally. The significance of the EEG abnormalities in these cases is always difficult to interpret.

Both of these cases, however, demonstrate the necessity of constant medical and otological supervision. The effects of the permanent nerve-sensory hearing loss, and the minimal brain "injury" are certainly compounded by the recurrent middle ear infections that increase the hearing deficit, and these add to the child's confusion, frustrations, and difficulties, as well as those of his parents and teachers.

The difficulties in assessing these children are apparent. Accurate hearing tests, in particular, are not easy to obtain by any single method, even at the ages of six to seven years. A special program can be arranged, however, for each child, based on his specific difficulties, even though a definite diagnosis cannot be made when the child is seen for the first time. Continued experimental and clinical research and clinicopathological studies will, in time, increase our knowledge of the disturbed mechanisms involved in these difficult cases.

SUMMARY.

1. The terms brain "injury" and impaired hearing are

defined. Children with cerebral palsy and mental retardation are not included.

2. The behavioral characteristics and the speech and language disorders in these children are outlined.

3. The experimental effects of sensory deprivation and brain stem lesions, and the known clinical effects of cortical lesions are described.

4. Essential details of the neuroanatomy of the auditory system, the results of recent neurophysiological experiments and clinical electroencephalographic studies are presented as a background for a deeper understanding of the problems involved in "brain-injured" children.

5. The details of a qualitative method of investigation and the comprehensive treatment of these cases are outlined.

6. Two illustrative cases are presented with comments on the problems involved.

CONCLUSION.

Many disciplines contribute to the understanding of human behavior, and each one has its own peculiar virtues and limitations.[8] There are many avenues to a meaningful knowledge and appreciation of the primary importance of the sense of hearing in the functions of the human brain, and modern otology has much to contribute to this fascinating subject.

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CARCINOMA OF EPIGLOTTIS. DIAGNOSIS AND SURGICAL TREATMENT.*†

Twenty-Five Year Survey.

WILLIAM A. LELL, M.D.,

Philadelphia, Pa.

The object of this paper is to review my personal experience with carcinoma of the larynx in general, but specifically to evaluate the knowledge gained in the diagnosis and treatment of 436 primary malignant lesions of the epiglottis that have come under my observation in the past 25 years. This material is derived from patients seen in my office, and in the Bronchologic Departments of various hospitals which are under my direct supervision, in addition to those patients seen and treated at the Graduate Hospital during my long period of association with the late Dr. Gabriel Tucker.

Any discussion of carcinoma of the epiglottis must of necessity also include some consideration of the larynx, since the epiglottis anatomically is a segmental portion of the rest of the larynx, and in an advanced lesion, it is sometimes difficult to ascertain the actual site of origin of the tumor. Because of this, a serious attempt was made to try to separate the lesions arising in the epiglottis proper from those arising in other areas of the larynx.

Anatomically, the detection of any abnormality in the epiglottis proper should not be difficult in the hands of a competent laryngologist, but unfortunately, many of these patients are not seen by a competent laryngologist in the early stage of their disease.

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†From Department of Broncho-Esophagology and Laryngeal Surgery, Graduate School of University of Pennsylvania, Philadelphia, Pa.

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Clinically, carcinoma of the epiglottis may be a very deceptive clinical entity, because, unlike lesions arising from the region of the vocal cords, it may not produce voice changes or acute distress until the disease becomes far advanced.

Growths arising in the area of the vocal cords, as a rule, produce varying degrees of voice changes which arouse the suspicion that something is wrong and causes the individual to seek medical aid to try to ascertain the cause of his voice changes.

On the contrary, lesions arising in the epiglottis proper may produce only a vague feeling of discomfort which is so often associated with the distress present with a sore throat, postnasal discharge, and chronic tonsillitis. Because of the persistence of the vague discomfort, many of these patients are considered neurotics and too often classed as suffering from globus hystericus syndrome.

In spite of repeated visits to physicians for aid for the relief of their discomfort, many of these patients were given various types of medication without any attempt at a thorough examination of the area from which the discomfort arose. Review of the histories of many patients reveals that, in spite of the fact that their symptoms were of long duration, during which time several physicians were consulted, only occasionally had anyone even put a tongue depressor in the back of the throat, let alone any attempt made at mirror laryngoscopy.

It is indeed a sad state of affairs that a structure so readily accessible to examination by such a simple procedure as mirror laryngoscopy should be so sadly neglected. It is particularly so since many of the far advanced cases could have been spared much misery and suffering if only the lesion had been detected in its early stage. I believe the time has come when mirror laryngoscopy should be a definite part of any complete examination of any patient with any symptoms of discomfort in the throat and adjacent structures.

Mirror laryngoscopy should not be considered a technique which belongs in the domain of the otolaryngologist alone, but rather as an indispensable procedure that can be utilized

by all segments of the medical profession with efficiency, with a little determination and effort. It is only by this means that we may be able to detect lesions early, and save many of these unfortunate patients the misery to which they would otherwise be doomed.

Mirror laryngoscopy should be made an obligatory part of the training of all medical students, so that this basic training

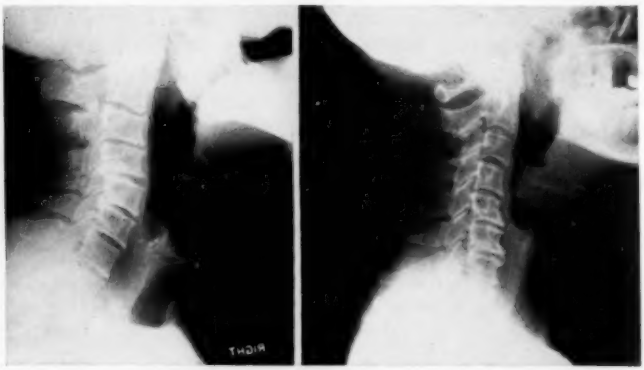

Fig. 1. Two different patients with different size lesions. The left early. The right far advanced.

could be further augmented during the subsequent periods of internship and residency. Is it possible that mirror laryngoscopy has lost its fascination because of its simplicity, or is it because we have not sufficiently stressed its importance? It is true that in certain patients, mirror laryngoscopy might be totally unsatisfactory because of anatomical changes in the structure and configuration of the epiglottis which prevents clear visualization of the glottis of the larynx and the under surface of the epiglottis. In these types of patients, we must resort to other types of aids. Lateral soft tissue films of the neck, centered over the larynx proper, may often give considerable detailed information of variations from the normal appearance of the laryngeal structures (see Fig. 1). Need-

less to say, in order for these studies to be helpful, they must be exact, so as to show soft tissue detail.

Ogura[1] and others have reported the value of laryngograms in detecting laryngeal abnormalities, and this technique should be utilized more frequently in cases that are difficult to visualize the larynx and adjacent structures by mirror laryngoscopy. Maloney,[2] Norris,[3] and others have stressed the importance of avoiding pitfalls in diagnosis of laryngeal disease and the need of detailed evaluation of suspected laryngeal abnormalities.

Necessarily, all of these preliminary means of getting added information are for the major purpose of guidance so that the greatest benefit may be gained during examination of the suspected area under direct laryngoscopy.

Direct laryngoscopy should be used without hesitation in all suspected laryngeal pathology both for thorough visual examination of the larynx, and whenever indicated to obtain tissue or secretions for accessory means to try to establish the underlying nature of the abnormality. I feel justified in saying that the successful treatment of any patient's disease depends primarily upon early diagnosis. Devising fancy operations for the eradication of hopelessly neglected conditions will never be the solution to man's ills. The solution lies in the early detection of the ill before it has gotten beyond control, so that the problem is no longer one of removing the growth from the patient, but rather the removal of the patient from the growth.

I am sure that if many of these patients had been more fortunate in early diagnosis, their names would not have appeared in the obituary column when they did.

In my experience, the following are indispensable aids in the early diagnosis of carcinoma of the epiglottis:

1. History:
   a. Local discomfort.
   b. Hoarseness.

2. Mirror laryngoscopy, Planography, Cine Fluoro-photography.

3. Routine Preliminary Studies:

a. General Medical Examination.

b. X-ray studies of the chest, swallowing function, and lateral films of the neck.

c. Serology and sputum examination.

d. Direct laryngoscopy and biopsy.

4. Pathology:

a. Relative frequency of types of involvement:

1. Squamous cell Carcinoma:	94%
2. Basal cell:	1%
3. Papillary:	2%
4. Adenocarcinoma:	1%
5. Sarcoma:	2%

5. Age:

a. Between 32-83 years.

b. Majority, 40-63 years.

6. Sex:

a. Predominant in men, 7-1.

Statistical Classification:

Total laryngeal tumors observed:	11,475
Benign lesions:	8,321
Malignant lesions:	3,154
Total Malignant Tumors of the Larynx:	3,154
Total Malignant Tumors of the Epiglottis:	436

Classification of Lesions Involving the Epiglottis:

Early:	87
Moderately advanced:	101
Advanced:	131
Hopeless:	117

Treatment and Results:

Early Group:	87
Laryngectomy:	57
Laryngectomy and radical neck	4

Refused operation:	26	
Irradiation:	22	
Sought aid elsewhere:	4	
Unilateral metastasis before laryngectomy:	4	
Metastatic glands after laryngectomy:	3 of 57 patients—	5.3%
Five-year survival 59.0%	36 of 61 patients	
Moderately Advanced Group:	101	
Laryngectomy:	66	
Laryngectomy and radical neck	7	
Refused operation:	28	
Irradiation:	21	
Sought aid elsewhere:	7	
Unilateral metastasis:	5	
Bilateral metastasis:	2	
Metastatic glands after laryngectomy:	12 of 66 patients—	17%
Five-year survival 46.5%	34 of 73 patients	
Advanced Group:	131	
Laryngectomy:	40	
Laryngectomy and unilateral radical neck:	13	
Laryngectomy and bilateral radical neck:	14	
Refused or inoperable:	64	
Irradiation:	43	
Sought aid elsewhere:	21	
Unilateral metastasis:	11 of 40 patients	37%
Bilateral metastasis:	4 of 40 patients	
Five-year survival 23.8%	16 of 67 patients	
Hopeless Advanced Group:	117	
Laryngectomy and unilateral neck:	9	
Laryngectomy and bilateral neck:	16	
Inoperable or refused operation:	92	

Irradiation: 67
Sought aid elsewhere: 25

Survival Rate of Hopeless Advanced Groups:
Laryngectomy and unilateral neck: 9
5 less than 9 months
1 Survived 9 months
1 Survived 14 months
2 Survived 2 years

Laryngectomy and bilateral neck: 16
12 Survived less than 1 year
1 Survived 12 months
1 Survived 16 months
2 Survived 18 months

Five-year survival rate: 0

DISCUSSION.

In order to avoid confusion, it might be worth while to discuss the results of treatment of each group individually. For clarification of the degree of involvement of the primary lesion, it was thought advisable to group patients into four groups, dependent upon the extensiveness of the lesion and the area of the epiglottis involved.

The early group comprised all of the cases in which the lesion was small and self-contained, and located in the central area of the laryngeal side or on the under surface of the epiglottis (see Fig. 2).

In the moderately advanced group were classified lesions in the similar area of the epiglottis, but which had extended out toward the brim but had not spilled over the brim into the outer surface (see Fig. 3).

The advanced group includes all the cases where the primary lesion had extended beyond the brim of the epiglottis, but was still confined to the epiglottic structure, but had not extended to or invaded the contiguous structures such as the

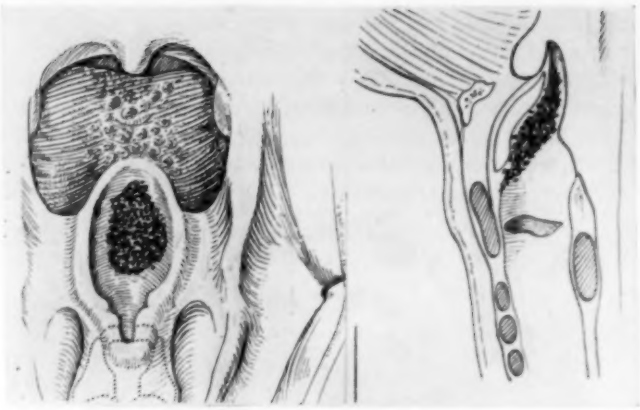

Fig. 2. Early group.

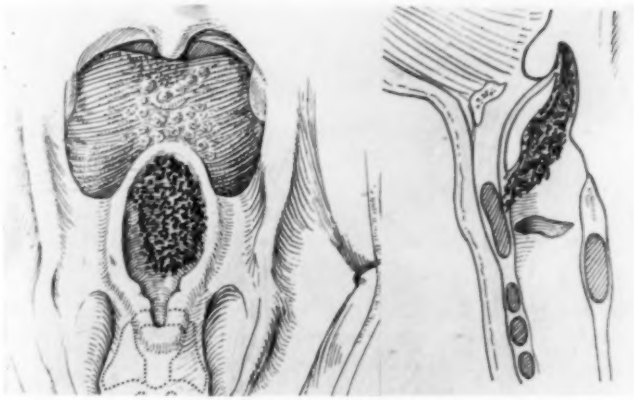

Fig. 3. Moderately advanced group.

base of the tongue, pharyngeal wall, pyriform sinuses or the posterior and subglottic regions of the larynx (see Fig. 4).

As the name implies, in the hopeless group were included all the extensive cases which, although first originated in the epiglottis, had subsequently invaded the adjoining structures to varying degrees of extensiveness. Needless to say, the

primary lesion was not the only problem for consideration in this group. Many of these patients, in addition to the presence of bilateral, hard, fixed nodes, were in extremely poor physical condition; some secondary to malnutrition because of dysphagia, others because of complicating systemic diseases. At best, treatment of this group was primarily aimed to relieve their discomfort and over-all palliation of the disease (see Fig. 5).

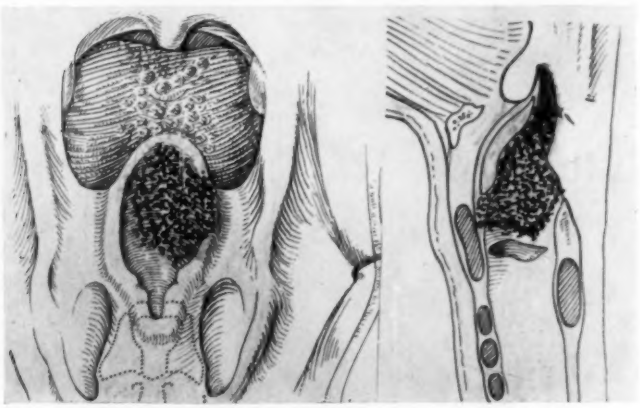

Fig. 4. Advanced group.

In reviewing these four groups, one fact becomes outstandingly apparent—the greater the extension of the lesion, the less the potential possibility of cure.

In the early group where the lesion had been detected and treated early, the survival rate was gratifying; however, with the progressive lag in the early detection and treatment of the lesion, the end results become more and more discouraging. In this series of cases, it was possible to obtain a 59 per cent, five-year cure in 36 of 61 patients by eradication of the primary lesion and the local metastatic extension, surgically. In the next group, where the primary lesion was moderately more advanced, the survival rate dropped to 46.5 per cent,

and only 34 of 73 patients survived five years. In the advanced group, where the lesion had extended beyond the confines of the epiglottis, there is a further drastic drop in the survival rate of 23.8 per cent, which again demonstrates that once the pathology has begun to get out of control, the chance of eradicating the disease becomes less, regardless of the type or number of surgical procedures utilized.

One is faced with total frustration in the futile attempt to try to eradicate the disease completely in the group that

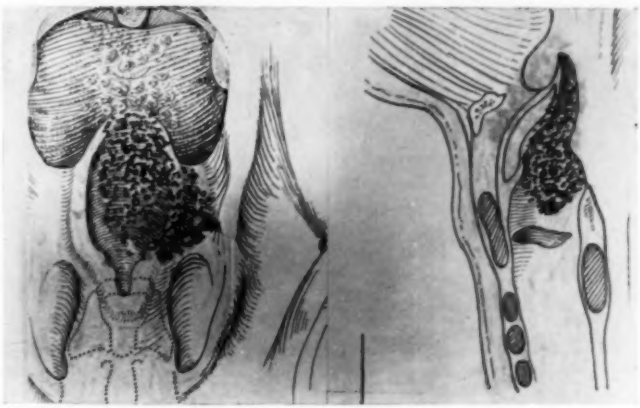

Fig. 5. Hopeless group.

has reached the hopeless stage. Despite the fact that 25 patients out of the group of 117 had various types of extensive surgery, only two survived 18 months, and only two survived two years. This indeed is discouraging.

Relative Merits of Laryngectomy and Simultaneous Radical Neck Dissection.

Reed,[4] Ogura,[5] O'Keefe,[6] Putney,[7] and others have favored a more radical approach in the surgical treatment of carcinoma of the larynx by advocating radical neck dissection on the basis of the size of the primary lesion even though the

patient had no palpable evidence of cervical adenopathy. This is advocated as a means of prophylaxis whereby the potential area of possible invasion is removed as a matter of precaution, thereby giving the patient a better chance for cure. This premise might be justified if the pattern by which cancer spreads were more uniform and predictable; however, since no one can foresee what the behavior of any of these lesions may be in any individual case, it would seem rather radical to carry out a formidable surgical procedure on the basis of anticipation that metastasis will take place but is not yet apparent. A classical radical neck dissection, if carried out with meticulous care, is quite an extensive procedure that should be used when definite palpable adenopathy is present, or in those cases where there is a doubt whether glands are palpable, or in individuals where follow-up might be undependable or unreliable.

Rouviere[8] in his extensive studies of the lymphatic distribution in the human body has clearly shown that there is a remarkable variation not only in the distribution but also in the abundance of the lymphatic channels in different individuals. This offers a sensible explanation of one of the many possible reasons why some individuals with extensive primary lesions never develop metastatic spread while others with minimal appearing growths may show evidence of extensive metastatic involvement not only to the neck but also to distant organs (see Fig. 6).

In view of the experience gained from the observation of the cases here reported, the greatest need of urgency is in the adequate and total removal of the primary lesion with associated neck dissection in those cases that show definite evidence of localized cervical metastasis. The total removal of the primary lesion by wide resection immediately eliminates any possible further primary metastatic spread than has already occurred. Consequently, nothing is lost and much may be gained by close periodic check-up of those patients to see whether any metastatic nodes become apparent. By this means several patients were saved the ordeal of the radical neck because it became evident that metastasis had extended

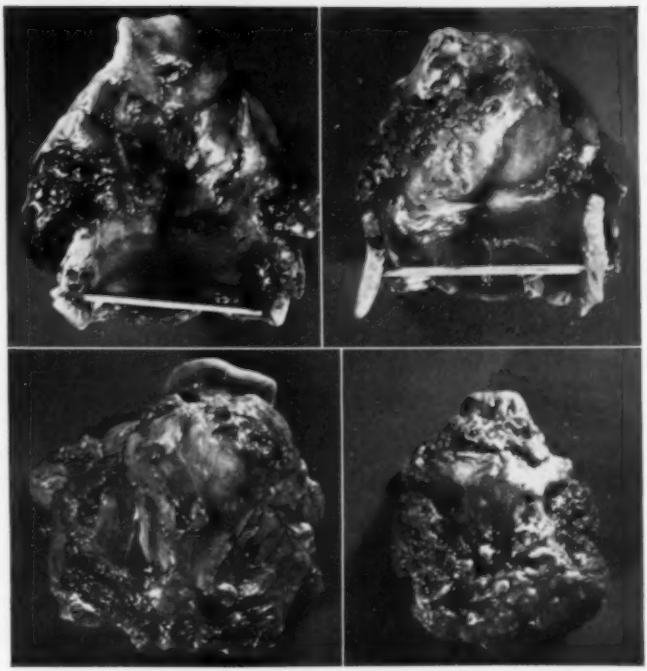

Fig. 6. Far advanced Ca of epiglottis removed by wide resection but no radical neck. Never developed any cervical metastasis.

far beyond the cervical region and certainly these patients would not have been helped by neck dissection; again there is also the possibility of metastasis to the contralateral side.

As mentioned, the difficulty of the close follow-up of a patient may be justification for doing a radical neck in those cases where there was strong indication that metastasis might occur because of the extensiveness of the lesion; however, I have been pleasantly surprised in some cases where no palpable cervical nodes were present, in spite of the extensiveness of the primary lesion, and none ever developed following laryngectomy. Though this is the exception, it still merits consideration.

The aim and purpose of any operative procedure should be to eradicate the growth with due consideration of the safety of the patient's life. Naturally, the greater the problem, the greater becomes the risk which one has to face. Within this same framework, one also should have the thought to preserve normal structures whenever possible, so as to restore the patient as closely as possible to his previous state of health, so that he may still have the desire to live. Although radical neck dissection does not present a serious threat to life when properly and skilfully done, it is associated with definite morbidity and potential complications.

I doubt that any of us would submit to an operative procedure unless we were definitely assured that it was necessary and that there was definite evidence for its indication rather than that it was being done on a presumptive basis because the lesion may have already spread to these structures. Again, I do not believe that enough serious consideration has been given to the fact that metastatic extension may also occur by way of the blood stream in an advanced lesion, in which case the spread would be beyond the lymphatic chain of defense. I am sure that many of us have seen such situations.

As might be expected, there is some variation in the results reported by different individuals as to potential incidence of metastatic involvement in carcinoma of the larynx treated by laryngectomy and elective neck dissection. Norris[3] reported six (37.5 per cent) out of 16 cases that microscopically showed evidence of positive nodes in which laryngectomy and elective neck dissection had been done simultaneously. Putney[7] found that in a group of 62 cases of laryngectomy and simultaneous elective neck dissection the incidence of positive nodes was 16 (26 per cent). O'Keefe[6] reports incidence of 23.5 per cent of positive nodes found in 68 patients in which laryngectomy and elective radical neck had been performed.

No direct comparison can be made of my results with the above findings, because of the supraglottic location of the lesion and the difference in the classification as to degree of involvement in my cases; however, in my early group there was no comparable high incidence of metastasis following

laryngectomy in the cases which had no palpable glands at the time of operation. In this group only three of 57 patients or 5.3 per cent developed subsequent metastatic cervical nodes; however, this figure jumped rapidly to 12 of 66 patients or 17 per cent in moderately advanced, and to 15 out of 40 patients, 37 per cent, in the advanced group. All the patients in the hopeless group already had either unilateral or bilateral metastasis, so they offered no choice.

One definitive lesson may be derived by the digestion of all these statistics, namely, that carcinoma of the larynx or of the epiglottis may be cured in most cases if the diagnosis is made sufficiently early.

SUMMARY.

A 25-year survey of carcinoma of the epiglottis reveals glowing evidence that the diagnosis is not made early in spite of the fact that this structure is easily accessible to examination. A great deal of this lag in diagnosis could be overcome by stimulating the interest of the medical profession in mirror laryngoscopy and more thorough examination of the patient's focal point of complaint.

Evaluation of the patients observed over this period reveals that 59 per cent of the cases of carcinoma of the epiglottis survived five years or longer, when the diagnosis was made early, and that the survival rate decreases in proportion to the lag in diagnosis. In the hopelessly advanced cases the longest survival period was only two years despite extensive surgical procedures. The cure of carcinoma, whether in the larynx or in any other part of the body, lies in early diagnosis.

The surgical eradication of the disease likewise is in direct proportion to early diagnosis.

There is urgent need of emphasis of the importance of the use of mirror laryngoscopy by all segments of the medical profession.

CONCLUSION.

In a 25-year survey of 436 cases of carcinoma of the epi-

glottis, the diagnosis was not made until late in over 60 per cent of the patients.

This lag in diagnosis was partly due to improper examination of the patient because of inadequate training or lack of interest in mirror laryngoscopy.

In 87 patients where the diagnosis was made early, it was possible to eradicate the disease surgically by laryngectomy in 57 cases, and by laryngectomy and radical neck in seven cases for a five-year survival rate of 59 per cent.

In the hopelessly far advanced lesions, in spite of the combined use of radical surgical procedures in 22 of 117 cases, only two survived two years.

Elective radical neck dissection as an adjunct to laryngectomy is not the answer to the surgical cure of carcinoma of the epiglottis. The best chance for cure lies in early diagnosis.

Consideration of elective radical neck dissection merits discriminate evaluation as to its indication.

One should not partake too deeply of infusions that may stimulate the ego to try to accomplish the supernatural.

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THE AREAL RATIO AND VARIATIONS IN NORMAL HEARING.*

WILLIAM H. SAUNDERS, M.D. (By Invitation),

Columbus, Ohio

The normal human ear, unaffected by disease, acoustic trauma, or the attrition of age, is known to be more efficient in some individuals than in others. A recent study[1] showed that when the hearing of 2,175 normal children was tested, there were wide variations in the acuity of hearing. All of the children tested had been examined by otologists and were said to have normal ears. There was no history of unusual exposure to noise or of familial hearing loss. A graph, adapted from the unpublished material describing this study, is given as Figure 1. The chart shows that at the particular frequency represented (1,000 cps.) there are wide variations in the levels of hearing acuity. Similar differences were found at other frequencies. These differences are well beyond the limit of error associated with audiometric testing. They represent real differences that are present in any large group of normal individuals.

What is the explanation? Are synapses along auditory pathways significantly different from one person to another? Does a variation in the number of auditory hair cells explain it? Are there important functional differences in the external or middle ear?

Unable to answer the problem with certainty, this paper will demonstrate that, at least theoretically, differences in hearing acuity can be explained by anatomical differences in the middle ear. Of course it has not been possible to correlate directly audiometric findings and postmortem anatomical measurements. One is not likely to have made audiometric

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measurements on a healthy young patient and then receive his temporal bones for a study a short time later. Rather, one must try to correlate differences in anatomical structure in one group with differences in hearing acuity in another group. If sufficiently large numbers of observations are included the method becomes statistically valid. Even if audiometric determinations could be correlated with postmortem findings in every case, one still could not say positively that

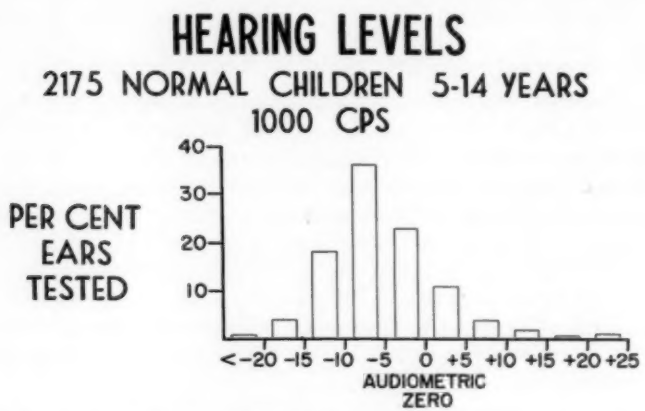

Fig. 1. Variations in hearing acuity among 2175 young normal individuals at 1000 cps. Adapted from E. L. Eagles, et al.: Report of Subcommittee on Hearing in Children of the Committee on Conservation of Hearing, Amer. Academy of Ophthal. and Otolaryngol., 1960. Published in Trans. Amer. Acad. Ophthal. and Otolaryngol., May-June, 1961.

other factors were not partially responsible for variations in hearing acuity.

To attempt to regain the energy lost (about 30 decibels) when air-borne sound pressure meets liquid in the cochlea, the middle ear has evolved as a mechanical transformer. By means of the lever ratio (the ratio between the long process of the malleus and the long process of the incus), the ear regains some 2.5 db. Then by virtue of the *areal ratio* (the ratio between the respective areas of the tympanic membrane and the footplate of the stapes), the ear regains another 23-25 db. The sum of the decibels gained by both the lever

action and the areal ratio is 25-27. This figure represents the approximate mechanical advantage afforded by the middle ear in attempting to regain the 30 db lost when sound pressure traveling in a gaseous medium (air) meets a liquid (perilymph). The remaining 3 to 5 db not restored by the mechanical action of the middle ear are apparently forever lost, unless, or until, evolutionary processes correct the situation.

Differences in the lever ratio from person to person are negligible. If these differences were to be made considerable, the length of one ossicle would become so great as to outgrow the middle ear. On the other hand, variations in the areal ratio may readily explain the observed differences. Minor deviations in the area of either the tympanic membrane or the stapedial footplate could alter the areal ratio significantly.

Materials and Technique.

Forty-three temporal bones from 43 different adult cadavers obtained at autopsy were dissected. At first an attempt was made to measure the tympanic membrane directly. Direct measurements are not practical. Because of the intimate attachment of the malleus to the drumhead, because the eardrum is conical, because the pars flaccida tears easily (and for other reasons), accurate direct measurement of the tympanic membrane is most difficult. A better method (and the one used here) is to remove the tympanic membrane and the annulus tympanicus from the tympanic sulcus and then to regard the tympanic sulcus, except where it is deficient superiorly, as representative of the circumference of the eardrum.

To prepare the specimen the cartilaginous and osseous parts of the external meatus are carefully removed with a rongeur or dental engine and bur until the entire eardrum and annulus are visible. There must be no overhanging walls of the external meatus to obscure vision. Next, the tympanic membrane and the annulus are removed. Then the malleus, incus and stapes are avulsed. This preparation exposes the

oval window and superimposes, just external to it, the tympanic sulcus.

With the anatomical preparation complete, the next step is to photograph each bone in the same way so that all specimens will be measured under the same conditions of magnification. Actually, because the object of the experiment is to *compare* two areas and not to measure either absolutely, it would not matter greatly if exact distances were not maintained from camera lens to specimen. Even if care were not taken to prevent distortion, the oval window would be enlarged or reduced in the same proportion as the tympanic sulcus since the two are so close and the distance from the camera lens so great. With a 35 mm. reflex camera, exposures were made on Kodachrome film. The specimens were placed so that both the oval window and the tympanic sulcus faced the lens directly—in other words, the largest possible area of each opening was measured. The result is that on each transparency are seen the margins of the oval window as well as the tympanic sulcus.

Projecting the transparency produces a greatly magnified image. Tracings are made on white paper of the two circumferences to be compared. These tracings show a large tympanic sulcus and near its center, a much smaller oval window.

The paper on which the tracings were made was weighed to make certain that each sheet, as well as different areas in each sheet, were of uniform weight. Finally, the areas traced were cut out and weighed on an analytical balance. After the outline of the tympanic sulcus (which included an outline of the pars flaccida) was weighed, the oval window outline was cut from the larger piece and weighed. The two weights were compared by dividing the weight of the piece of paper representing the tympanic membrane by the weight of the piece of paper representing the oval window. This figure represented the areal ratio—the actual, not the effective, ratio.

This method does not measure the actual size of either the eardrum or the footplate. It merely establishes a ratio between the two. To check on the accuracy of measurements

and to guard against the possibility that one circumference, being slightly closer to the camera than the other, might be enlarged to a significantly greater degree, pieces of ruled millimeter paper were photographed under similar conditions. No significant distortions were found; but again, even if there had been distortions, the discrepancy would have been equal from ear to ear and *differences* in area ratio, the only measurement considered here, would have remained the same.

Results.

Areal ratios as determined by the above method in 43 ears taken from 43 individuals varied from a low figure of 14.5

Areal Ratios as Computed from 43 Temporal Bones Taken at Autopsy from 43 Cadavers.

18.9	18.5	20.8
19.7	23.5	14.5
18.3	21.3	20.3
18.1	19.0	20.5
26.1	20.1	17.2
17.5	26.4	17.0
22.0	21.5	22.3
28.0	23.2	34.4
16.5	16.6	24.4
24.8	20.7	16.3
20.6	20.3	18.6
24.4	20.0	18.8
19.4	23.0	18.6
17.5	22.0	18.8
26.8		

Fig. 2.

to a high of 34.4. The mean figure was 20.3 and the average 20.8. The entire list of measurements is given in Figure 2.

DISCUSSION.

The effective area of the tympanic membrane is not the same as its actual area because the eardrum does not work as a whole. The most peripheral parts of the drum move less than its central part. The effective area, therefore, will always be less than the actual area. The effective area of the eardrum is figured by Wever and Lawrence[2] as being about two-thirds of the actual area. Accordingly the effective areal ratio is no greater than two-thirds of the actual ratio.

According to Wever and Lawrence only a few measurements of the areal ratio in human ears have been made. They, themselves, measured both ears of a single individual and obtained ratios of 18.2 and 19.1. Fumagalli is quoted as having examined one ear with a ratio of 21. Békésy "obtained a value of 26.6, again probably in a single ear." In four cat ears Wever and Lawrence obtained values of 32.7, 33.5, 37.3, and 42.6. They do not say whether these ears were taken from four cats, three cats, or two cats. Whittle is quoted as measuring the areal ratio in eight guinea pig ears with the values: 26.1, 26.2, 27.6, 27.8, 27.8, 28.3, 30.4, and 30.9. Wever and Lawrence conclude, "It is plain that there are large differences among species and among ears of any one species."

CONCLUSIONS.

In the human, the areal ratio varies widely from one individual to another.

If, as is generally accepted, the areal ratio is the most important part of the transformer action of the middle ear—acting to recover some 25-27 of the 30 db of energy lost when sound pressure passes from air to water—then the variations in the areal ratio as found here may explain, at least in part, the known differences in the hearing acuity of young normal individuals.

SUMMARY.

The areal ratio was measured in each of 43 ears taken from 43 human cadavers. The observed differences are great enough to explain, at least in part, the known variations in the hearing acuity of young normal individuals.

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A FIVE-YEAR REPORT ON FENESTRATION OF THE OVAL WINDOW WITH VEIN GRAFT.*

JOHN J. SHEA, M.D.,

Memphis, Tennessee.

Fenestration of the oval window was first reported at the original Symposium on Mobilization of the Stapes in Montreal in May, 1956.[1] In this first operation the entire stapes was removed and the oval window covered with a thin slice of subcutaneous tissue. The sound conducting mechanism of the middle ear was rebuilt with a Teflon replica of the stapes. This first patient's hearing was improved, and her bothersome tinnitus reduced.

Because of the possibility that some epithelium might be included in the slice of subcutaneous tissue, and other disadvantages, the search was continued for a better material with which to cover the oval window. In July, 1957, I began using the vein graft after seeing a general surgical colleague replace a thrombosed artery with a free vein graft.[2]

From the beginning the patient's own posterior crus, if it was intact after removal of the footplate, was used to rebuild the sound conducting mechanism of the middle ear because of the difficulty of making a replica of the stapes from the hard and yet brittle Teflon; but, all too often, the posterior crus was not long or straight enough to make good contact with the vein graft invaginated into the oval window, and, furthermore, it was often fragile, poorly nourished, and completely replaced by otosclerotic bone so that its permanence was very doubtful. Because of these difficulties with the Teflon replica of the stapes and the patient's own posterior crus, in August 1957 I began to use lengths of polyethylene "90" tubing to rebuild the sound conducting mechanism of

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the middle ear. Not only was polyethylene nearly reaction-free in the middle ear, it could also be quite easily cut into the desired lengths and shapes at the time of surgery, and, quite fortuitously, in most cases the size "90" tubing would fit snugly onto the lenticular process to make a secure connection with the incus.

Results with the operation continued to improve as the technique evolved, and by May 1958 at the second Symposium on Mobilization of the Stapes in San Francisco, I was able to report 89 operations with good hearing improvement in half, and no dead ears.[3] The greatest limitation of the operation at that time was the thickened, rigidly fixed footplate with otosclerotic narrowing or obliteration of the oval window niche. Such a footplate could not be completely removed, and, in some, only a small opening could be made in the oval window. There was usually some hearing improvement after surgery, but in three to six months the opening in the oval window narrowed or closed, and the hearing returned to the preoperative level.

With the introduction to this surgery of the Wullstein contra-angle hand piece,[4] and later the slowly turning, vibration-free stapes drill,[4] the entire stapes could be removed in every case. In addition, the excess bone mounding up on the promontory and other margins of the oval window could be removed, to saucerize the opening and thin its edges so as to follow that cardinal principle in fenestration of the oval window so thoroughly proven in fenestration of the horizontal semicircular canal. At once there was a sudden increase in the immediate hearing improvement, both as to the extent of the hearing gain in each case, and the percentage in which this gain could be accomplished; but, more important, the long-term results were also much improved, with 90 per cent of those operated having a closure of the air-bone gap to within 10 decibels, which gain has been maintained, with remarkably little decrease, to the present time.

It is interesting that the level of 90 per cent closure of the air-bone gap was reached at once, as soon as the entire stapes was removed, and has been maintained with little improve-

ment, despite various refinements in the technique, during the last three years. In addition, results of 90 per cent closure of the air-bone gap to within 10 decibels have been reported by others, from various parts of the world, whenever the entire stapes has been removed, no matter what method was used to rebuild the sound conducting mechanism of the middle ear.

There is naturally some later proliferation of the bone of the margins of the oval window when the entire footplate is removed, and the fenestra is saucerized; but if the opening made is large enough, then the brief proliferation that does occur postoperatively will not be enough to reduce significantly the size of the opening and the effectiveness of the operation. Presumably, the stimulus that causes this great excess bone formation on the margins of the oval window is metabolic, and in most patients the time for this growth has long since passed when surgery is done; so removing this excess bone will be followed by the natural proliferation and healing of bone that follows injury to it at any time but will not be followed by the return of this entire excess.

In addition, when the vein is invaginated into the oval window slightly, it seems to retard this postoperative proliferation of bone and brings about healing of the raw edges more rapidly. It has already been suggested by Tonndorf[5] that the inverted parabola shape of the invaginated vein in the oval window has advantages for sound conduction. It was observed by Sooy[6] in a series of experimental operations on monkeys that there was active new bone formation at the edges of the oval window covered with gelfoam, but much less in those covered by vein; but it is difficult to conclude from these experiments that gelfoam supports further proliferation of bone and vein retards it.

It should be noted that there is a distinct difference between the patient with a swollen, doughy, white footplate, not firmly fixed in the oval window, with a well-preserved joint space, which does very well after surgery, and the patient in whom the otosclerosis is widespread and invasive, obliterating the margins of the oval window, sometimes with

otosclerosis mounding up to the level of the promontory and facial nerve. Such an oval window is more difficult to open, the hearing result is not so good, and the danger of further hearing loss is greater; but, despite these limitations, the results fully justify operating on this group of patients unless the otosclerosis is too massive.

Unhappily, with the use of the drill in the oval window there began to occur with greater frequency, the occasional patient with further, or even total, hearing loss following surgery. The complete cause of these further hearing losses is not always apparent, but enough is known of them to take precautionary measures to reduce them to a minimum. Although the number of patients with further, or even total, hearing loss increased with the introduction of the oval window drill, the good results soared at the same time, and I am convinced that the results do not justify doing the operation unless the drill is used in the oval window and the entire footplate removed, despite the occasional patient made worse by surgery.

After two years the original evolution of the operation was complete, and during the last three years the technique of fenestration of the oval window with vein graft has remained with very little change. The operation begins by fracturing the arch of the stapes, removing the mucoperiosteum from the circumference of the oval window for a distance of about 1 millimeter, and controlling the bleeding that invariably occurs. If the footplate mobilizes in the attempt to fracture the arch, the stapes is not removed until the mucoperiosteum is stripped from the circumference of the oval window and the bleeding controlled; otherwise, blood will get into the vestibule. This is not particularly harmful in itself, and can be removed by aspirating in the vestibule, at once, with a 24 gauge needle, but it may disturb the patient and provoke vomiting. With the footplate in place, the excess bone mounding up on the margins of the oval window, especially on the promontory and anterior rim, is carefully removed with a small cutting burr, turning slowly, to saucerize the oval window and prepare a concave bed for the placement of the vein graft. The mucoperiosteum must also be removed from

the facial nerve. If there is otosclerosis on, or beneath, the bone of the Fallopian canal, it can be safely removed with a small diamond burr, turning slowly. This has been done many times with no injury to the underlying nerve. If the footplate is mobilized in the attempt to fracture the arch, the stapes is now removed intact by tilting it to one side until an edge comes out of the oval window and the airtight seal in the vestibule is broken. If, when the arch of the stapes has been removed, the footplate remains in the oval window, it is divided with a triangular perforating burr, cutting from the facial nerve to the promontory. The two halves of the footplate are then removed with a short right-angle fistule hook.

In widespread invasive otosclerosis, the joint space between the footplate and the oval window will be obliterated, and in such a patient the fenestra must be slowly created in the oval window by saucerizing the edges and drilling out the footplate. This is best begun with small cutting burrs, 2/0 and 4/0. After the oval window has been saucerized and the footplate thinned, the opening into the vestibule is best made with a diamond burr, wet with blood or perilymph, turning slowly, so that it will not throw bone chips into the vestibule.

The vein used should be thin, but not too thin, preferably taken from the back of the hand below the wrist so that there will be no muscle fibers in it. The vein is further thinned in a special vein press, which also eliminates the vein's natural tendency to curl, so that it can be put into place more easily, and can be invaginated into the oval window slightly. Sooy has demonstrated that pressing the vein does not harm the elastic layer, which is the important one to preserve.[6]

In most cases a 4 millimeter length of polyethylene "90" tubing, beveled at its lower end, is used to rebuild the sound conducting mechanism. This usually makes a firm connection with the incus by pressing it onto the lenticular process.

Sometimes the lenticular process is deflected away from the oval window, or is too small, or entirely missing. When this occurs the polyethylene "90" tubing, one-half millimeter longer, with two notches in the upper end is used. This is pulled up onto the lower end of the incus to make a secure

connection with it, despite the deficiency of the lenticular process. If the lenticular process is missing and the lower end of the incus is somewhat atrophic, it is best to use a length of polyethylene "90" tubing, 1 millimeter longer than usual, with two holes near the top. The lower end of the incus is then fitted into these two holes.

If the patient has had a prior unsuccessful fenestration of the lateral semicircular canal, even if the hearing is somewhat improved by the operation, and the fenestra in the horizontal canal is still partially open, it is possible to do fenestration of the oval window in the usual way. The sound conducting mechanism is rebuilt with a longer length of polyethylene "90" tubing, usually about 6 millimeters long, the top of which is wedged beneath the under surface of the drum just behind the malleus, being held in position by stuffing the cut end of the chorda tympani nerve (cut posteriorly at the *iter chordi posticus*) in the top of the tube. This prevents the tube from slipping loose later, or perforating the drum by pressure necrosis, and makes a sufficiently secure connection to the drum so as to rebuild the sound conducting mechanism very efficiently.

It is surprising how well these patients have done, with closure of the air-bone gap, in half of those operated (17 out of 34), despite the loss of the lever mechanism of the normal ossicular chain.

It is true that there is some proliferation of bone after fenestration of the oval window in which considerable removal of bone is done at the margins of the oval window with the drill, because while these patients usually make a good hearing gain which is sustained later, they do not have the complete return to normal hearing usually seen when the otosclerosis, as seen grossly, is confined to the footplate; nevertheless, as stated previously, I favor drilling away the otosclerotic bone surrounding the oval window, and those who are doing no more than picking out a piece of the footplate and covering the small opening created with some amorphous materials such as gelfoam, fat, or connective tissue, will find that bony closure will eventually occur, and the

long-term hearing results in these patients will not be good. I would caution against the use of gelfoam as the only covering of the oval window after removal of all or part of the stapes. The membrane that usually forms over the oval window may be quite thin and can rupture with sudden barometric changes, or Politzerization of the ear, or even spontaneously. The use of this gelfoam may be followed by purulent labyrinthitis and meningitis from an acute upper respiratory infection, coming on without provocation as late as two years after being put into place, as occurred in one case recently. It is easier to cover the opening in the oval window with gelfoam than vein, but I do not believe that the results will be as good, nor that the hazard to the patient's hearing and life is justified.

One precautionary note about the use of prophylactic antibiotics. From the beginning I have used some wide-spectrum antibiotic prophylactically, and for several years I have had no postoperative infections. During three months this Winter I did not give any prophylactic antibiotics, and I had two patients with purulent otitis media coming on several days postoperatively. Once again I have returned to the prophylactic antibiotics, starting early the morning of surgery, to get an adequate blood level in the patient at the time of operation, so that the blood that accumulates in the middle ear and external canal will contain antibiotic also. It is interesting that this acute otitis media coming on several days postoperatively was not complicated by labyrinthitis or meningitis, and did not prevent the patients' getting a good hearing gain, which is proof of the effectiveness of the vein as a seal for the oval window.

As for the stapedectomy and wire-fat technique introduced by Schuknecht at the second Symposium on Mobilization of the Stapes in 1958[3] and the wire-vein plug technique as introduced by Kos in 1960,[7] I have not done these operations, so I have no experiences to report.

RESULTS.

An examination of the results of a large series of opera-

tions is always complicated by the lack of reliable follow-up in some patients, and the variations in technique by which the operation is done while the series is being collected.

The hearing improvement in the first patient operated on five years ago has been maintained, and it is interesting to note that her entire stapes was removed.

The 89 patients, reported at the second Symposium on Mobilization of the Stapes, in which the entire footplate was not always removed, have remained about the same as they were three years ago, with a good hearing improvement in half, and no dead ears.

Of 1,396 patients operated on during the last three years in which one and in some cases two-year follow-ups are available, and in which the entire footplate was always removed, 1,277 or 91 per cent have closed the air-bone gap to within 10 decibels. Breaking these results down by bone conduction groups the success rate is as follows:

Group A—714 patients 90 per cent.
Group B—434 patients 94 per cent.
Group C—176 patients 91 per cent.
Group D— 72 patients 89 per cent.

It is interesting that the percentage of success is slightly lower than average in Group A cases, and this is probably because this group contains more younger patients who do not do so well with this surgery, as will be demonstrated later. This overall success rate of 91 per cent compares with the 94 per cent success rate at three months in a like series of patients reported previously.[8]

It is very gratifying that the success rate dropped only from 94 per cent to 91 per cent from the three-month to the one-year test, and in those in which two-year tests are available there is no further loss from the one-year test. It would appear that there is a slight attrition (3 per cent) during the first year, but after this occurs the healing is complete, and no further loss takes place.

Most significant of all is the analysis of results by age

groups. Out of 1,295 patients, grouped by ages, the success rate, as measured by closure of the air-bone gap to within 10 decibels, was:

12 per cent of 9 patients, age 11 to 20.
25 per cent of 59 patients, age 21 to 30.
Approximately 90 per cent of the rest, age 31 to 80.

It is apparent that the younger patients do not do so well with this surgery, and perhaps it would be best to defer operation on them until the fourth decade of life, when the metabolic activity of the otosclerotic bone is less.

Fifty per cent of 34 patients who had fenestration of the oval window after prior unsuccessful fenestration of the horizontal semicircular canal had a successful result.

Repeat fenestration of the oval window was done on 22 patients with a good result in nine, or 40 per cent.

A drillout of massive, widespread, invasive otosclerosis was done in 89 patients, with success in 78, or 89 per cent.

By comparison, anterior crurotomy was done on 271 patients, with good results in 182, or 67 per cent.

Further hearing loss has been very consistent in about 2 per cent, with complete loss in 0.5 per cent. This further hearing loss has usually occurred in the difficult drillout case, where the cochlear function was already depressed, especially where prior unsuccessful operation had been done, but occasionally it occurred in a simple, uncomplicated case for no apparent reason.

COMPLICATIONS.

The complication most feared is, of course, further total hearing loss following surgery. As stated above, the complete cause of these further hearing losses is not always apparent, but the greatest cause seems to be excessive trauma at the time of surgery from too much drilling in the oval window when the otosclerosis is of the widespread type. It is interesting that the complications of further hearing losses tended to occur more often in those patients in whom there was

massive otosclerosis, requiring considerable drilling away of bone. These were the patients with decreased cochlear function (the average of the three speech frequencies for bone conduction was often between 25 and 40 decibels) and they, quite clearly, were the least suitable for this or any type of surgery. It has been argued by the opponents of this type of surgery that these patients should not be operated upon in the oval window, and that they be reserved for fenestration of the horizontal semicircular canal. It is worthwhile, however, to point out that most of these patients, with massive widespread otosclerosis in which the greatest percentage of complications occurred, unfortunately have poor bone conduction and would not be suitable candidates for fenestration of the horizontal semicircular canal at all, and it remains for these patients to have oval window surgery or nothing at all. For that reason, despite the fact the complication of further hearing loss is greater in these patients with massive otosclerosis in which considerable drilling has to be done, the results in these patients, in most cases, still justify attempting the operation.

An analysis of the complications and failure in patients by age groups indicates the young patients in whom the otosclerosis began in the first or second decade of life have poorer results with surgery, and have many more complications. In deciding whether or not surgery should be attempted, it is well to consider whether the patient's hearing loss began quite early in life, whether the otosclerosis is massive and widespread, and the bone conduction is reduced. If all three factors are present, such a patient is best not operated upon because these three contribute to making the operation more difficult and the likelihood of further hearing loss more probable.

SUMMARY.

The evolution of the technique of fenestration of the oval window is presented. The turning point was the complete removal of the footplate and saucerizing the oval window which resulted in closure of the air-bone gap to within

10 decibels in about 90 per cent operated upon, which results have remained very much the same to the present time.

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NEWS RELEASE.

The Section on Otolaryngology of the Southern Medical Association will meet in Dallas, Texas, on November 6-9, 1961.

The first day will feature a television surgical clinic on Laryngectomy, and in the afternoon, Texas Day, papers on: Surgical Approaches to the Nasopharynx; Tympanoplasty; Scleral Buckling; Prostheses in Middle Ear Surgery.

Tuesday will feature papers covering: Branchial Cysts and Fistulas; Helpful Hints in Nasal Surgery; Control of Tonsillar and Adenoidal Bleeding by Electro-coagulation and Fluothane Anesthesia; Vein Grafts in Otological Surgery; Technique of Laryngectomy; So-called Sphenopalatine Ganglion Neuralgia; Fungal Infections of the External Ear Canal.

For additional information please contact the secretary, Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, West Virginia.

DECOMPRESSION OF THE FACIAL NERVE IN EXPERIMENTAL BELL'S PALSY.*†

FRANCIS H. MCGOVERN, M.D.,
Danville, Virginia,

and

J. S. HANSEL, M.D. (By Invitation),
Charlottesville, Virginia.

The value of the decompression operation in cases of complete Bell's palsy has been a subject of considerable controversy and even ridicule.[1] In spite of the favorable clinical evidence advanced by surgeons of high international reputation, many physicians remain skeptical of the surgical treatment of Bell's palsy; indeed, some even deprecate the modern methods of medical management of this disease.

The attitude regarding the decompression operation is due to a number of reasons. In a disease which will recover spontaneously in a month or more, or with suitable treatment in a matter of a few weeks, in 80 to 90 per cent of the cases, the clinician is reluctant to mention the possibility of surgical intervention. Even in those cases of severe paralysis, when the symptoms, the clinical observations and the electrical reactions indicate a nerve degeneration rather than a simple conductive block, the patient is often hopefully advised to await regeneration of the nerve and chance the possibility of incomplete recovery rather than undergo a debatable operation. Then, too, experienced otologists are not in agreement as to the time and indications for the operation. If the nerve is decompressed there is no available way of knowing whether improvement will result or whether the outcome will be superior to the non-operative treatment, because incomplete

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†From the Department of Otolaryngology, University of Virginia Hospital, Charlottesville, Va.

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recovery is often found in the cases operated upon after the usual two months' waiting period.

As Sullivan[2] has pointed out, if attention is paid to the truly unfortunate 10 to 15 per cent of individuals who do not recover at all or only in part, more than casual interest and laissez-faire therapy will result. The prevention of the permanent facial disfigurement of partially recovered peripheral facial paralysis is a major problem in otology. The experiments here reported were done in an endeavor to obtain additional evidence of the value of the decompression operation in the treatment of Bell's palsy.

Bell's palsy is regarded as a peripheral facial nerve paralysis for which no cause can be found. The process is thought to be an edematous neuropathy. As postulated by Hilger[3] and others, the edema of the facial nerve is presumed to be due to a series of vascular changes following a segmental arteriospasm of the perineural vessels supplying the nerve in individuals possessing a labile autonomic nervous system. The paralysis results from the edema of the primary ischemia and the secondary compression and vascular stasis of a swollen nerve enclosed in a bony canal. According to Cawthorne[4] and to Kettel[5] the major area of edema is located in the vertical segment of the nerve in the region of the stylomastoid foramen, although Williams[6] found that the swelling and ecchymosis of the nerve sheath at times may extend to the tympanic portion of the nerve.

The factors of a nerve coursing through a rigid bony canal and the singular arrangement of its blood supply are thought to be of paramount importance in the vulnerability of the facial nerve to paralysis. Cawthorne has emphasized the uniqueness of the facial nerve. Because it passes through a longer bony canal than any other nerve and can be involved readily in adjacent temporal bone diseases and injuries, it is paralyzed more than any motor nerve in the body. In our experiments we were ever amazed at the abrupt narrowing of the nerve as it enters the canal, and the exceedingly strong fibrous collar constricting the nerve at the stylomastoid for-

amen. This anatomic situation is the basis for the surgical treatment of Bell's palsy.

The sequence of events following the initial vascular insult has been well discussed by Collier.[7] If the edema is of modest degree the resultant ischemic paralysis is a temporary conduction block which affects only the myelin sheath without damage to the axons. The electrical reactions remain unchanged, and the recovery in this instance is usually rapid and complete. When the vasospasm is greater or long continued, or when the Fallopian canal is small in size or with a tightly constricting stylomastoid collar, the subsequent increased anoxia produced by secondary compression of the nerve results in a degenerative lesion involving the axis cylinders and the supporting structures. Recovery takes place on regeneration of the nerve but with the inevitable sequelae following re-innervation: imperfect muscle function, asymmetry, contractures, and associated movements. Prolonged ischemia is followed by complete nerve atrophy.

The surgical problem facing the otologist is to separate those cases of Bell's palsy in which a degenerative process is likely to occur from a simple conductive lesion. At the present time there is no precise way of foretelling which case will proceed to nerve degeneration or which case will recover spontaneously. The theoretic optimal time for surgical intervention is before nerve degeneration takes place, within hours or days following the onset of the paralysis.

Because there is no exact way of knowing which case of complete Bell's palsy will proceed to nerve degeneration or when a reversible lesion exists, otologists have viewed the problem of decompression as follows: Sullivan[2] states, "A Bell's palsy presenting complete facial paralysis, loss of the faradic response, accompanied by pain, is indicative of a severe degenerative lesion and warrants immediate surgical interference." Cawthorne[4] advises decompression in cases of complete paralysis of a month's duration when there is no response to electrical stimulation. In his opinion the purpose of the operation is to promote a quicker recovery of improved function in the cases in which full recovery is unlikely. Kettel,[5]

on the other hand, has little faith in the electrical tests and bases his indications for the operation on his wide clinical experience in facial paralysis. Except in cases of complete paralysis accompanied at the onset by considerable pain in which emergency decompression is recommended, he advises surgery, if after two months of observation there are no signs of spontaneous movement. Martin[8] agrees with Kettel's opinion. There is general agreement that severe pain is a significant symptom and is often followed by imperfect recoveries. Korkis[9] states that the operation is justifiable, "if, after adequate and early medical treatment, there has been no recovery in three to five weeks, and especially if electromyography indicates a severe lesion." Williams[6] advises decompression of the nerve if there is no sign of beginning regeneration, as indicated by electromyography, after two months. His conservative approach is designed to avoid early surgery yet with the knowledge that the delay will affect facial disfigurement in only 2 to 3 per cent of his patients.

The gross and microscopic observations of the nerve as seen at operation have uniformly been described as swelling, reddening and ecchymosis of the nerve sheath. Ballance and Duel first recorded the swollen appearance of the nerve, although Collier[7] has advised caution in assessing this finding because the nerve normally fills the canal during life, and the nerve bundles bulge out when the sheath is opened. We have not made this observation in our work on dogs. Under magnification Cawthorne[4] found an abnormal constriction of the nerve at the level of the stylomastoid foramen with a swollen nerve extending 5 to 10 millimeters tapering upwards. He noted one or more thin hemorrhagic streaks running for 2 millimeters above the site of constriction and one or more pink tinged patches in the swollen segment of the nerve. In cases of long standing the nerve was reduced to a shrunken strand. In Williams'[6] experience the swelling and reddening of the nerve were not confined to the region of the stylomastoid foramen but extended to the post-tympanic portion of the nerve. Sullivan[2] observed the nerve bundles in the affected nerve bulge when the pressure of the constricting perineural sheath was released by slitting the sheath. Kettel[5] found pro-

nounced edema of the nerve proximal to the constricted stylomastoid area in 80 out of 136 cases; he has observed no evidence of bulging of the normal nerve when the sheath is opened. In long-standing cases of Bell's palsy the nerve was atrophic.

Because of the rare opportunity of obtaining specimens of the facial nerve during an episode of Bell's palsy, histological reports are unusual. Both Jongkees[10] and Kettel[5] have reported on the microscopic appearance of the chorda tympani nerve obtained at the time of the decompression operation for Bell's palsy. In Jongkee's case the nerve was found to be free of inflammation. The myelin sheaths were very swollen and the axis cylinders showed nerve degeneration. In Kettel's case the perineurium was normal. The capillaries of the nerve were dilated, and the whole nerve was edematous. The myelin sheaths were degenerated, and the axis cylinders were almost completely lacking.

Kettel[5] states that five reports have been published of postmortem examination of the facial nerve of patients with peripheral facial palsy who died of various other causes. The descriptions of the microscopic pictures of these cases are much alike. They presented degeneration of the myelin sheaths and axis cylinders but no inflammation. The myelin sheaths were decomposed into globules and droplets. When the axis cylinders had not completely disappeared, the remaining part showed marked signs of degeneration.

There is scant reference in the current literature on experimental investigations related to the cause and treatment of Bell's palsy. Experimental peripheral nerve injuries and experiments related to the compression of the median nerve in the carpal tunnel have shown that ischemia resulting from constriction of the nerve is followed by edema proximal to the lesion. Denny-Brown's[11] experiments indicate that the damaging effect of pressure on nerves is related to occlusion of the blood vessels to the nerve. He cites the demonstration of the enormous pressures oxygenated frog nerves can withstand without impairment of conduction.

In a comprehensive article on the etiology and treatment of Bell's palsy, Sullivan and Smith[2] reported their experiments designed to study the blood vascular supply of the facial nerve and to observe the nerve and other changes following paralysis induced by cold and by constriction of the nerve with a suture. They presented a method of experimentation in rats simulating the changes believed to exist in Bell's palsy.

The paralysis induced in the rats by cold was transient as shown by a temporary loss of response to faradism; there was microscopic evidence of some fluid collection in the nerves and an increase in vascularity of the vessels of the epineurium down into the perineurium.

The paralysis and loss of faradism induced by the suture lasted for hours or days depending upon the tightness of the suture. There was gross edema of the nerve extending from the suture to the stylomastoid foramen. Microscopically the nerves showed myelin degeneration with droplet formation and the axis cylinders showed fragmentation. The suture caused a constriction and produced death of the axons proximal to the obstruction and distal to it. Osmic acid stain revealed myelin breaking down to form droplets in the proximal segment with a more pronounced breakdown in the peripheral segment.

In 1952 and 1953 Coassolo[12] published three papers dealing with experimental studies on the facial nerve. He examined the electrical excitability of the facial nerve after refrigeration in nonsensitized rabbits and in rabbits sensitized with horse serum; only the sensitized rabbits showed evidence of nerve insufficiency. In the nonsensitized animals there were no macro or microscopic changes seen in the refrigerated nerves, whereas in the sensitized group the refrigerated nerve appeared grossly enlarged and of a reddish color, and the histologic study showed extensive changes: edema, infiltration of white blood cells, blood extravasations and myelin fragmentation and absorption.

Coassolo interpreted these findings as confirming the allergic hypothesis of "cold" facial paralysis rather than the

theory of an edematous vascular neuropathy. He concluded that the compression invoked by the bony wall of the Fallopian canal is not a factor in this type of facial paralysis.

To further confirm the allergic pathogenesis of Bell's palsy, Coassolo repeated the sensitization experiment and injected procaine intravenously before the application of ice to the nerve. The procaine was used on the premise that it inhibits the formation of histamine and alters the allergic mechanism. In the animals treated with procaine "cold" paralysis did not occur; the control animals developed the typical syndrome of paralysis.

The idea of our study was to induce a temporary bilateral facial paralysis in the experimental animal (dogs) and to observe the recovery rate of the paralysis after decompressing the Fallopian canal on one side, using the opposite side as a control.

Preliminary work on 12 dogs showed the paralysis obtained by freezing the exposed facial nerve trunk with a refrigerant, with or without protein shock, too transient to carry out the purpose of the experiment. Although we were aware of the objections to the constricting suture method of simulating Bell's palsy, this method provided the practical answer to the aims of the experiment and our conclusions can be judged accordingly. This preliminary study also showed that the decompression operation alone did not produce a detectable paralysis, although undoubtedly a varying amount of trauma was incurred.

The decompression and neurolysis of the facial nerve was accomplished chiefly in the region of stylomastoid foramen. The nerve was uncapped in its lower vertical segment, and the tight fibrous foramen area was opened widely from above downward. Because of the dense connective tissue sheath surrounding the nerve at the foramen, attempts to decompress the nerve by dissecting into the stylomastoid foramen from below resulted in destruction of the nerve. Sullivan[2] noted the dense compact periosteal sheath surrounding the

Rate of Recovery.

Dog	2 wks.		4 wks.		6 wks.		8 wks.		10 wks.		12 wks.		14 wks.		16 wks.		18 wks.	
	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.	rt.	lt.
1	4	4	2	0	0	0	0	0	0	0								
2	4	4	2	2	0	0	0	0	0	0								
3	3	3	3	1	2	0	2	0	2	0								
4	4	3	4	2	3	2	2	2	1	1								
5	2	2	0	1	0	0	0	0	0	0								
6	4	4	4	4	3	3	1	0	—	—								
7	4	4	3	3	3	1	3	0	—	—								
8	4	4	3	3	2	2	1	0	0	0								
9	3	3	3	3	3	3	3	3	3	0								
10	4	4	3	3	2	0	1	0	—	—								
11	4	3	3	1	3	1	3	1	—	—								
12	4	4	4	4	4	3	4	3	4	3	3	0						
13	3	3	0	2	1	0	0	0	—	—								
14	2	3	0	2	0	2	0	2	0	1								
Average	3.50	3.43	2.43	2.21	1.85	1.21	1.44	.79	1.11	.55								
15	4	4	4	4	4	3	4	3	4	3	4	3	4	3	3	1	3	0
16	4	4	4	4	4	4	4	4	4	3	3	3	2	2				
17	4	4	3	3	3	3	2	3	2	3	1	2	0	2				
18	4	4	3	4	2	3												
19	4	4	3	3														
20	4	4																
Average	4	4	3.4	3.6	3.3	3.3	3.3	3.3	3.3	3	2.6	2.6	2	2.3				

Right—ligature alone.
Left—ligature and decompression.

Fig. 1.

nerve just within and at the exit from the stylomastoid foramen in humans.

The paralysis obtained by tying the nerve trunk near the stylomastoid foramen with catgut lasts from six to ten weeks. The ties were applied in approximately the same location on the nerve, in as similar a manner and amount of pressure as possible. A .00 double ligature was used.

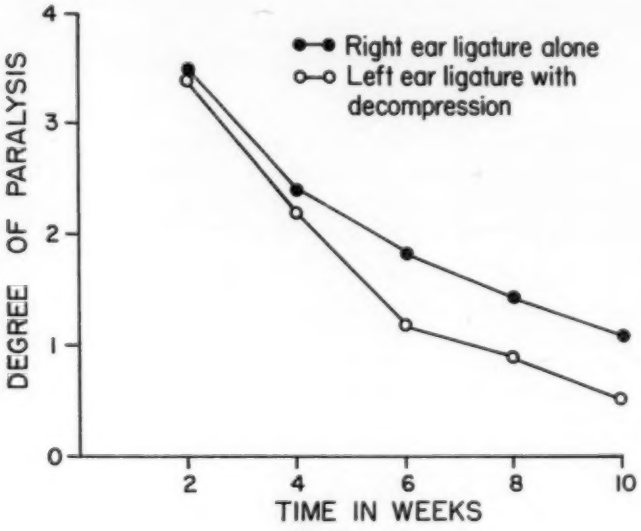

Fig. 2.

The dogs were observed periodically, recording the rate of return of facial nerve function, using the lid reflex as the index of re-innervation (see Fig. 1). The degree of movement was evaluated on the basis of 0 to 4—the latter indicating entire inaction and the zero indicating complete return of function.

RESULTS.

Twenty-five dogs were operated upon; of these three died before the completion of the experiment, and two dogs were

sacrificed to obtain histologic specimens after two and four weeks of their paralysis. Figure 2 is a composite graph showing the degree of paralysis and rate of recovery of the first 14 dogs. Although there was some individual variation, the graph indicates a faster rate of recovery on the side having the decompression. With three exceptions the nerves in the

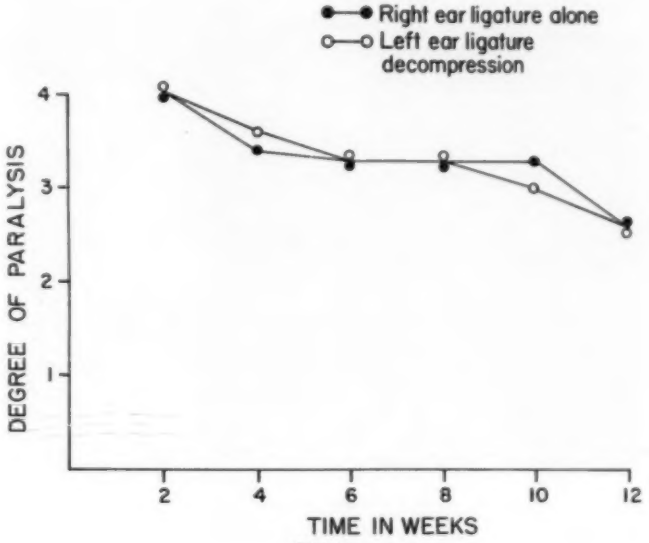

Fig. 3.

first group recovered two to four weeks sooner on the decompressed side than on the opposite side.

Figure 3 is a composite graph of the results of six uncompleted experiments. Five of the six dogs still show significant bilateral paralysis, and three of these are showing a faster recovery on the side not decompressed. We are unable to explain at this time why the recovery is prolonged in this group and why the opposite nerve appears to be recovering first. We presume an enlargement of surgical exposure in

these dogs for the purpose of photography, in which the regional blood vessels were ligated, to be a factor.

Through the courtesy of Dr. David E. Smith and Dr. Rodrigo Diaz-Perez of the Department of Pathology of the University of Virginia Hospital, six sets of nerves were examined microscopically. The nerves were removed two, four, eight (2), and ten (2) weeks postoperatively. A detailed

Fig. 4.

report of the findings will be published later. Their report is as follows:

"The study of the cases submitted demonstrates a variable picture of changes: The most constant were in relation to different systems:

a. Vascular.

b. Nervous.

c. Connective tissue.

Vascular Changes. Marked congestion in the nerves be-

tween the nerve fibers and also in the surrounding connective tissue.

Foci of hemorrhage, mostly located in zones adjacent to the nerves and perineurium.

Nervous Changes. Proximal to the ligature, in most cases, there was an area of edema. In the ligature itself, and distal

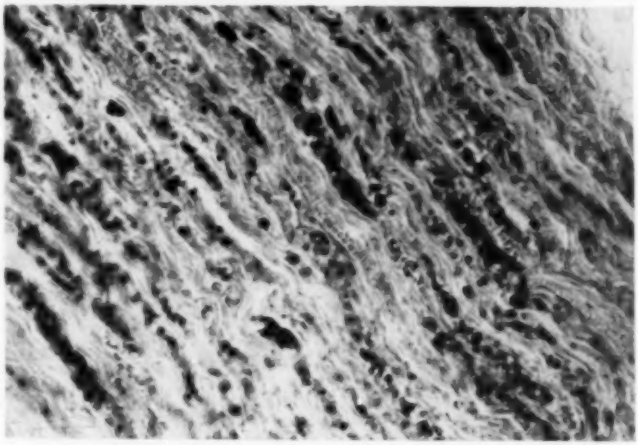

Fig. 5.

to the ligature there was a loss of nervous tissue. Also distal to the ligature there were areas of demonstrable globular swelling of the myelin sheath, and a great deal of fat droplets in the nerve fibers consistent with myelin sheath degeneration. In some cases the proximal area also shows these changes for a short distance.

Connective Tissue. Condensation of the fibrous tissue in the area of ligature.

These nerves show the reactions of severe acute compression and damage beneath the ligatures and Wallerian degeneration in the distal segment commensurate with the duration of the dog's life after ligature. Perhaps the most important obser-

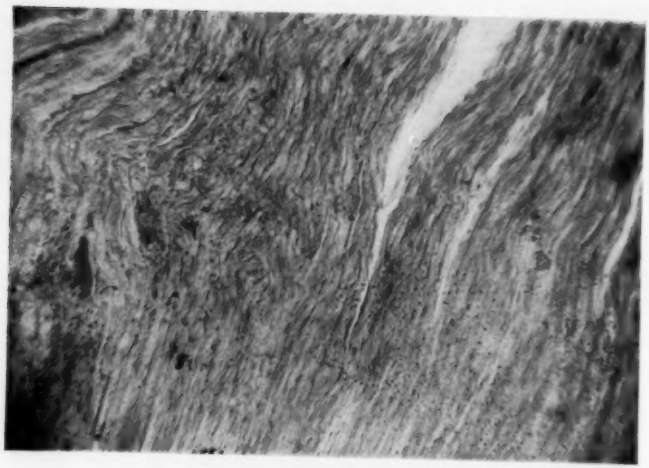

Fig. 6.

Fig. 7.

vation is that there are no appreciable qualitative or quantitative differences between the left and right nerve in any single animal, although there is considerable variation between various animals."

Figure 4 (Spielmeyer's stain) shows the portion of the nerve constricted between two catgut ligatures. The area proximal to the ligature shows edema; between the ligature and in the distal area there is a loss of nervous tissue and myelin sheath degeneration.

Figure 5 shows blood vessels with stasis and areas of globular degeneration in the myelin sheath.

Figure 6 shows edema of the proximal region; no visible nervous structure can be seen.

Figure 7 shows beginning globular swelling of myelin sheath degeneration.

COMMENT.

The significant anatomical feature observed was the narrowing of the nerve as it enters the facial canal. Indeed often the main trunk and branches measured 7 mm. in width, narrowing to 4-5 mm. at the foramen and to 2-3 mm. in the canal.

The constriction of the stylomastoid collar has been mentioned. Sullivan[2] has emphasized this area as the site of greatest edematous swelling in Bell's palsy, as seen under the dissecting microscope at operation.

Although edema is a significant feature of the microscopic picture, we did not note macroscopic swelling when the nerve was re-exposed for biopsy. The histologic findings in these dogs are similar to the published reports of nerves obtained from cases of Bell's palsy and from experimental work on facial paralysis.

In spite of the additional trauma inflicted upon the facial nerve by the decompression procedure, the decompressed facial nerve appeared to recover sooner than the nerve on the non-decompressed side.

We feel that our observations and results further corroborate the premise that the facial canal plays a fundamental part in the causation of Bell's palsy.

CONCLUSION.

A review of the current concepts of the etiology of Bell's palsy and the role of surgery in the treatment of Bell's palsy are presented. In an attempt to assess further the value of the decompression operation, a series of experiments was performed. In dogs a temporary paralysis was achieved by ligature compression of each facial nerve. In addition the nerve was decompressed in the region of the stylomastoid foramen on one side. It was found that the facial nerve tended to recover sooner on the decompressed side.

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A REVIEW OF AUDIOLOGIC FINDINGS AMONG PATIENTS WITH CEREBELLOPONTINE ANGLE TUMORS.*†

RICHARD M. FLOWER, Ph.D. (By Invitation),
and
RICHARD VIEHWEG, M.A. (By Invitation),
San Francisco, Calif.

In recent years, audiologists have become increasingly interested in the auditory sequelae of retrocochlear lesions and in the development of special techniques designed to differentiate these disorders from other pathologies with similar symptomatology. Academically, cerebellopontine angle tumors are of particular interest because of their relative specificity in locus and extent. Clinically, they are of obvious significance because of the gravity of the pathology and the need for accurate early identification.

The literature contains considerable variation in the terminology used to denote cerebellopontine angle tumors. Preference for particular terms is based upon varying concepts of their nature and origin. Pool and Pava[12] list several of the commonly used terms: perineural fibroblastoma, neurofibroma, fibroma of the VIIIth nerve, acoustic neuroma, acoustic neurinoma, lemmoma, lemmoblastoma, acoustic tumor, and Schwannoma, as well as cerebellopontine angle tumor. Dix and Hallpike,[5] however, differentiate between tumors arising from the VIIIth nerve itself (acoustic neuroma) and other tumors arising within or invading the cerebellopontine angle producing a secondary VIIIth nerve affection.

Hearing impairment, often combined with tinnitus, is the most common and earliest symptom of cerebellopontine angle

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†From the Audiology and Speech Clinic, Division of Otorhinolaryngology, University of California School of Medicine, San Francisco, Calif.

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tumors. It is reported in all but one of 145 patients, by Revilla[13]; in 292 of 300 patients, by Lundborg[11]; and in 112 of 122 patients, by Pool and Pava.[12] Since the auditory symptoms are usually the first to appear, patients with cerebellopontine angle tumors in the early stages of development are much more likely to be referred to an otologist, and subsequently to an audiologist, than to a neurologist or neurosurgeon.

The clinical audiologist encounters many problems in assisting in the identification of cerebellopontine angle tumors. One set of problems results from the relative rarity of these tumors. The characteristic audiometric patterns and test behavior of patients with more common conditions such as otosclerosis, presbycusis, and, to a lesser extent Ménière's disease, are reasonably well known and can usually be identified by an experienced clinician; however, the only widely recognized characteristic of hearing impairments resulting from cerebellopontine angle tumors is that, except in a very few reported instances, they are unilateral. The audiometric configuration of impairments attributable to these tumors remains as yet substantially undescribed. As a result, confusion with other retrocochlear disorders or even certain end-organ pathologies may occur.

Other problems occur in the interpretation of special tests, notably tests which seek to demonstrate the presence or absence of loudness recruitment. Wide variation exists in the reported clinical applications of specific tests, to the point of describing instances of findings of complete recruitment among patients with confirmed VIIIth nerve tumors. Many reports tend to treat recruitment as an all-or-none phenomenon, seemingly a serious over-simplification. Findings of so-called "partial" or "incomplete" recruitment as opposed to "hyper" or "over" recruitment are difficult, if not impossible, to interpret. Although none of the techniques described to date can be considered clinically infallible, particularly if used singly, tests of recruitment should unquestionably be utilized whenever possible and appropriate. With respect to the specific population under consideration here, these tests

have a serious limitation in that they are applicable only during the initial stages of the hearing impairment, since total loss in the affected ear frequently occurs relatively early in the natural history of the tumor.

Many very practical problems are often encountered by a clinician in attempting the assessment of the auditory disorders found among these patients. These problems are particularly common in a hospital audiology program. Patients hospitalized with suspected brain tumors are frequently very ill. Understandably, they may be anxious and apprehensive and unable to cooperate in long, critical listening situations.

During the past five years 22 patients with subsequently surgically confirmed cerebellopontine angle tumors have been seen in the Audiology and Speech Clinic of the University of California San Francisco Medical Center. As the result of some of the problems mentioned earlier, test results are in certain instances incomplete; nonetheless, in spite of some variation, some observations may be made regarding patterns of impairment in thresholds for pure tones, results of speech discrimination testing, and results of tests for loudness recruitment.

PATTERNS OF IMPAIRMENT IN THRESHOLDS FOR PURE TONES.

Although virtually all writers in the literature on cerebellopontine angle tumors refer to the attendant auditory impairment, references to specific audiometric patterns and presentations of actual findings are relatively rare. When audiometric findings are presented, they bear a striking similarity (Hallberg, *et al.*,[7] Kristensen,[10] Denny,[2] and Lundborg[11]). Despite this fact, it is frequently inferred that there is little similarity of pattern to be found among pure-tone audiograms of these patients.

A review of the findings on the 22 patients in this series indicates the following types of audiometric curves (see Fig. 1): seven with high frequency losses (patients R. S., I. R., C. S., G. J., R. H., W. A., and K. B.); two with gradually falling losses of mild-moderate degree (patients S. W.

and J. C.); six with flat impairments of moderate-severe degree (patients G. S., M. M., R. C., G. M., I. W., and E. S.); and six with total impairments on the affected side. One patient (I. R.) had an unusual trough-type curve; however, essentially normal thresholds were maintained through 500 cps.

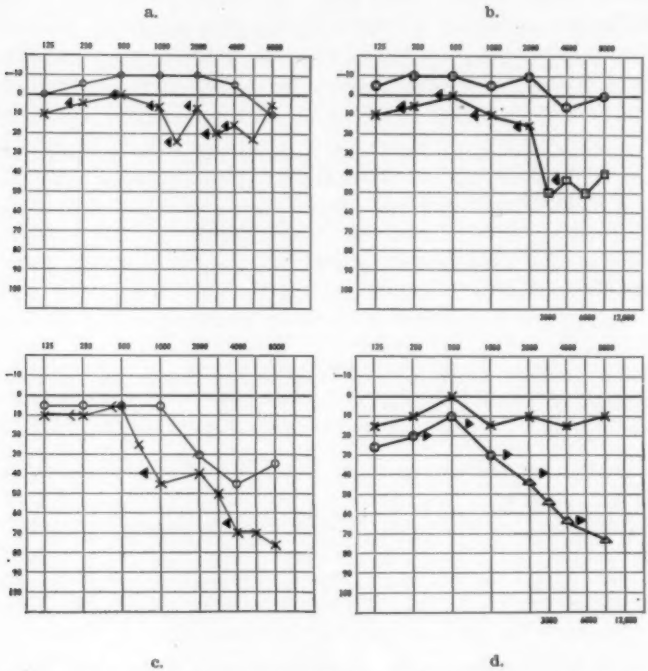

Fig. 1. Initial audiograms obtained on 16 patients with cerebellopontine angle tumors. a.—Patient R. S. (male, aged 21). b.—Patient I. R. (female, aged 36). c.—Patient C. S. (male, aged 51). d.—Patient G. J. (female, aged 68).

Although some individual variation occurs, it seems that a roughly characteristic audiometric pattern may be described, with the seemingly wide differences in threshold levels between certain patients attributable to the fact that tests have been conducted at different stages in the course of growth of the

neoplasm. Specifically, it is our hypothesis that the impairment usually begins with a very minimal depression of thresholds which would commonly be regarded as insignificant (see first audiogram in Fig. 2), progressing to mild to moderate high tone loss, increasing to invade the middle and low fre-

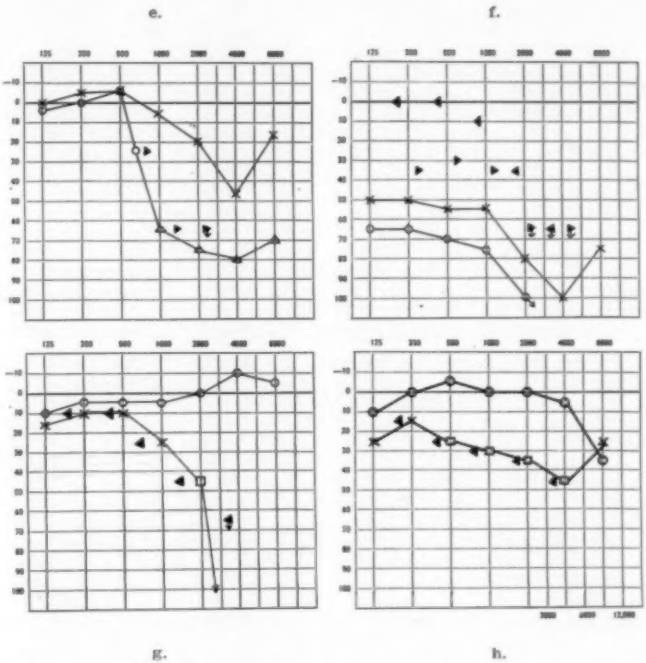

Fig. 1. e.—Patient R. H. (male, aged 53). f.—Patient W. A. (male, aged 59). g.—Patient K. B. (male, aged 22). h.—Patient S. W. (female, aged 63).

quencies, ultimately producing a flattened curve, and eventually resulting in a total impairment.

The literature contains frequent references to a statement made by Eggston and Wolff,[6] apparently based on observations by Haberman, Rhese, and Sieberman and Bengold, that the auditory components of cerebellopontine angle tumors are first evident in the depression of thresholds for the lower

frequencies with near-normal thresholds in the higher frequencies. In our patient series, we do not find a single instance in which the lower frequencies seem to have been the first affected; in all instances where the low frequency thresholds are reduced, the higher frequencies are equally or more impaired. In certain instances the thresholds at 8000 cps.

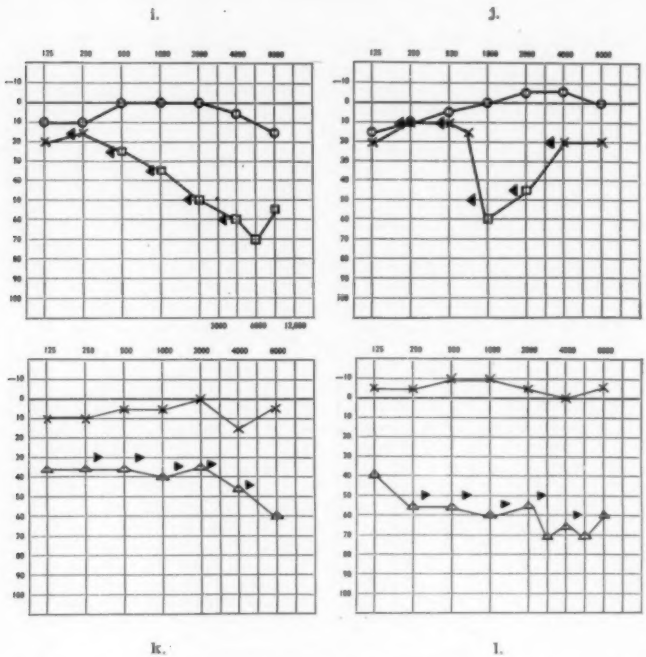

Fig. 1. i.—Patient J. C. (male, aged 28). j.—Patient D. P. (female, aged 28). k.—Patient G. S. (male, aged 51). l.—Patient M. M. (female, aged 51).

may be better than those at 2000, 3000, 4000, or 6000 cps. (see Fig 1; patients R. S., S. W. and D. P.), but with two exceptions this may be as much a comment on the effectiveness of masking at 8000 cps. as a real observation of sensitivity differences.

Support for the hypothesis regarding the progressive pat-

tern of decreasing sensitivity for pure tones may be found in the results of repeated testing on four patients in this series. In all other instances where repeated testing was possible, surgical or radiologic intervention had occurred between tests.

Patient K. B. (see Fig. 2) seems to exemplify the almost subclinical characteristics of the early auditory symptoma-

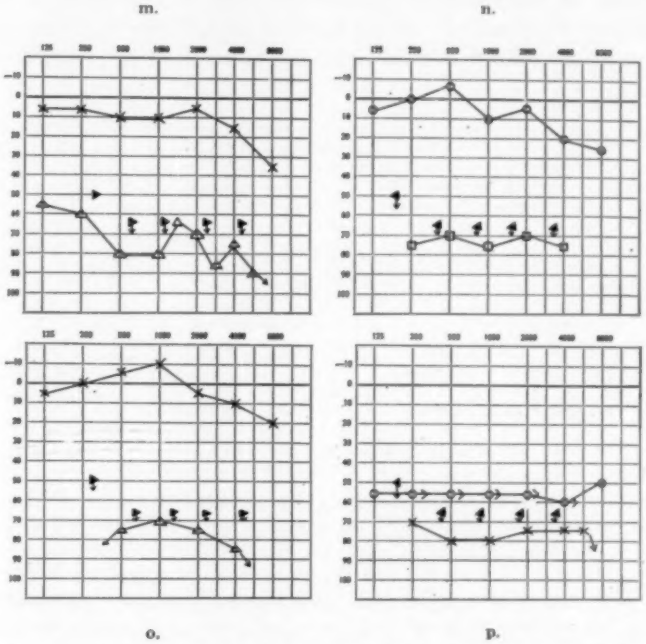

Fig. 1. m.—Patient R. C. (female, aged 52). n.—Patient G. M. (female, aged 59). o.—Patient I. W. (female, aged 48). p.—Patient E. S. (male, aged 38).

tology. At the time the first audiogram was obtained, the patient had consulted an otologist with the complaint that speech seemed "garbled" in the left ear. As shown in the first audiogram, a very minimal depression in pure-tone thresholds was evident in this ear. A speech reception threshold of 11 db and a discrimination score of 100 per cent were

obtained. Ten months later, the patient returned to the otologist complaining of an increase in the distortion of speech in the left ear and constant pain on the left side of his head. The otologist found an inactive caloric response on the left, at the second visit. The patient was referred to our clinic where the second audiogram was obtained. After this relatively short period of time, complete absence of sensitivity to tones at maximum audiometric output was evident in the higher frequencies. Even more striking was the patient's inability to discriminate anything more than the number of

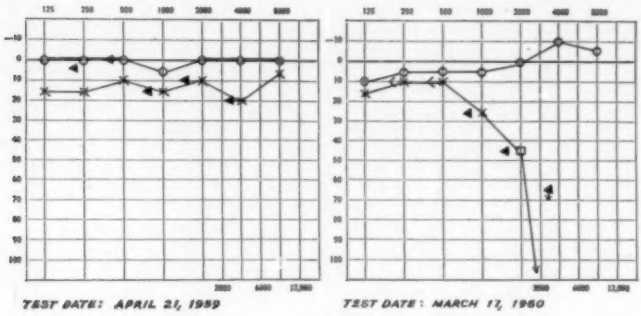

Fig. 2. Two audiograms obtained at a ten-month interval on patient K. B.

syllables in a stimulus word in the affected ear even at maximum output of the speech audiometer, although he was aware of speech at a level of 14 db.

Our experience with this patient prompts us to speculate on the observation of Dix and Hallpike[5] and other writers that "normal hearing" is sometimes found among these patients. A more accurate statement may be that during the early stages of the growth of the tumor, thresholds for pure tones within normal limits may be demonstrated, but that subtle changes in audition may be present which do not produce striking reduction in sensitivity for pure tones or even reduced speech discrimination.

Patient G. J. (see Fig. 3) seems to exemplify a patient in whom the original moderate high frequency loss progressed

during a five-month period until responses to the highest frequencies were absent, with a slight additional broadening of the impairment in the middle frequencies.

Patient G. M. (see Fig. 1) had been aware of a gradually progressive unilateral hearing loss of several years' duration. At the time the first audiogram was obtained a severe, flat, left ear impairment was evident. Subsequent testing three years later demonstrated total impairment on the left with an increase in the number and severity of other symptoms.

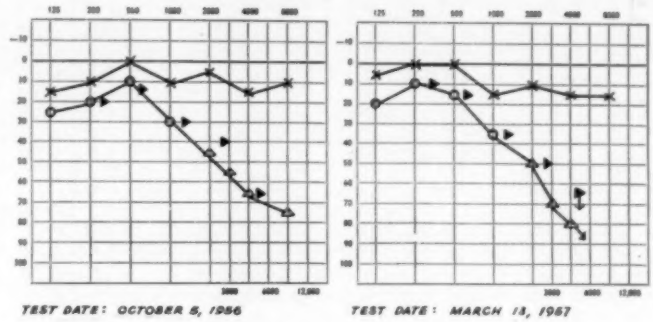

Fig. 3. Two audiograms obtained at a five-month interval on patient G. J.

Additional support for the progressive pattern of hearing impairment hypothesized earlier can be seen in Figure 4, a series of audiograms obtained on patient C. S. over a five-year period. The hearing impairment progressed from one originally characterized by a mild, gradually falling audiometric curve to total impairment on the affected side.

The true test of the postulated pattern of progressive impairment would be the establishment of a correlation between the degree and nature of impairment with other indices (such as onset, size, exact location, and rate of growth) of the point in the progression of the tumor at which these audiograms were obtained. Unfortunately, this is virtually impossible. Estimates of the point of the testing in temporal relationship to reports of the initial onset of symptoms are of little value

since a patient's awareness of onset varies considerably according to his sensitivity to relatively minimal changes in audition. Even if it were possible to establish the time of onset, the rate of tumor growth obviously varies greatly among these patients. The surgeon's description of the size of the tumor may give a rough indication of its history, but

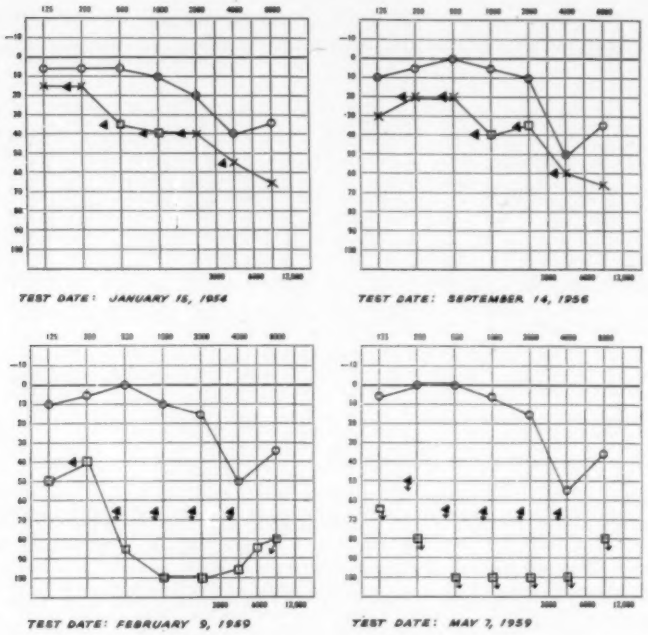

Fig. 4. Four audiograms obtained over a five-year period on patient C. S.

the degree of impingement on the acoustic nerve can only be conjectured. Any attempt at establishing relationships between test findings and estimates of time of onset or the extent of the tumor would, therefore, appear of dubious value.

In clinical practice, the audiometric identification of the initial onset of reduced auditory sensitivity may be extremely difficult because of pre-existing hearing losses of other etiol-

ogy. This situation is readily illustrated by patient W. A. (see Fig. 1) who had a long-standing mixed hearing loss prior to the probable onset of the tumor. More subtle interferences to early recognition can be observed in other audiograms in our series. In many instances, reduced thresholds for high frequency tones are evident in the unaffected ear,

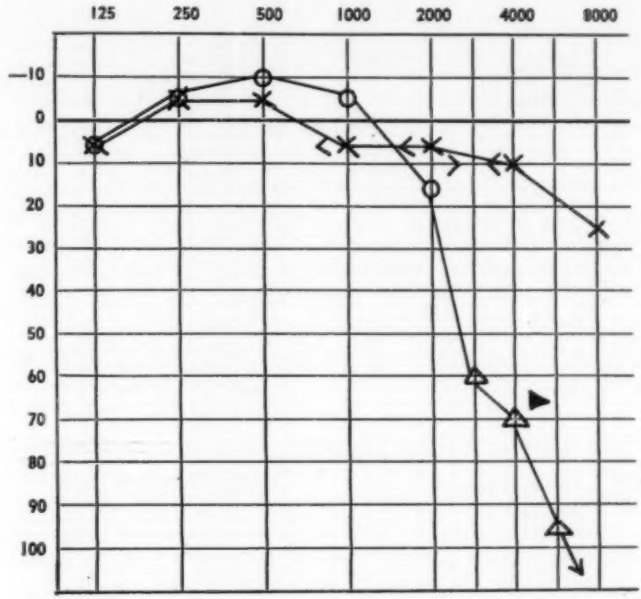

Fig. 5. Audiogram obtained on D. F., a patient with a diagnosis of Ménière's disease.

presumably as the result of noise exposure, presbycusis, etc. Assuming that a similar reduction in high frequency thresholds was present in the affected ear prior to the emergence of the tumor, the early high frequency impairment attributable to the tumor may not have been particularly striking, conceivably delaying investigation until it became more pronounced.

As mentioned elsewhere in the literature, even the charac-

teristic unilaterality of hearing impairment, usually considered to be the hallmark of the auditory symptoms of cerebellopontine angle tumors, is not an infallible diagnostic clue. A bilateral impairment was demonstrated by patient E. S. (see Fig. 1). In this instance we may be guilty of some generosity in the application of the term "cerebellopontine angle tumor"

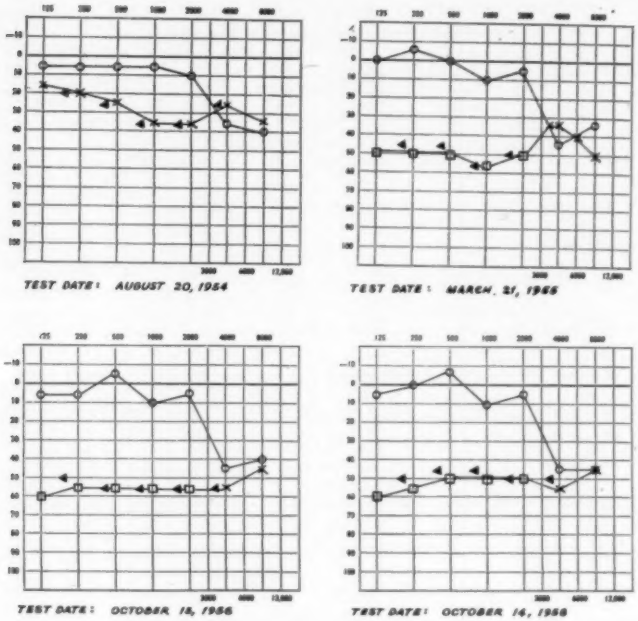

Fig. 6. Four audiograms obtained over a four-year period on A. F., a patient with a diagnosis of Ménière's disease.

since this patient had neurofibromatosis. Two neurofibromas were found, however, to be impinging on the VIIIth nerves bilaterally.

The audiometric pattern ascribed to cerebellopontine angle tumors, both during their initial stages and as they progress, although somewhat unusual, cannot be considered unique to this pathology. A pattern similar to the one found during

the early stages of a tumor is illustrated by patient D. F. (see Fig. 5) who had a unilateral high frequency impairment, tinnitus, and vertigo. An articulation score of 88 per cent was achieved, and recruitment was demonstrated in the affected ear. On the basis of a complete evaluation of the patient's symptoms, the diagnosis of Ménière's disease was made. Subsequent treatment based upon this diagnosis eliminated the vertigo and tinnitus, although the hearing loss remained.

A progressive pattern of impairment similar to that hypothesized for cerebellopontine angle tumors was illustrated by patient A. F. (see Fig. 6) who also had Ménière's disease. Since some reduction of thresholds for the lower frequencies was already apparent at the time the first test was administered, we cannot be certain that the higher frequencies were the first to be impaired; nonetheless, a gradually falling, then flattening curve, is clearly shown. A notable difference from the tumor pattern is evident, however, in that complete absence of responses to pure tones did not occur in the later stages of the course of progression; the loss apparently remained stable for over three years. Complete loss in an ear as the result of Ménière's disease is extremely rare in our experience.

As a result of our observations, we would emphasize the need for careful evaluation of the patient who may complain of distortion of speech or other qualitative differences in an ear which appears essentially normal by the routine audiologic testing battery. More sensitive measures should probably be added to the battery in these instances at the time of the initial evaluation. Procedures such as the distorted speech test described by Jerger[9] may prove useful. In any event, routine follow-up of patients experiencing distortion in an apparently "normal" ear should be recommended. Further, we believe a unilateral high frequency loss to be sufficiently unusual that its presence might well suggest the need for careful investigation and follow-up, since this pattern has been demonstrated, in several instances, to be the initial stage of the auditory impairment resulting from tumors at the cerebellopontine angle. Finally, we would stress the need for careful periodic follow-up of all known cerebellopontine angle

tumors in an effort to clarify further the hypothetical pattern of progression described earlier.

RESULTS OF DISCRIMINATION TESTING.

It has been observed that one of the major characteristics of the auditory deficits of patients with cerebellopontine angle tumors is the reduction of discrimination for complex sound stimuli which is inordinate when compared with the pure-tone audiogram. Jerger[8] in a recent article refers to the "bottleneck principle," stating, ". . . the process of transmitting the informational content of a complex auditory stimulus (*i.e.*, speech) encounters a very real bottleneck in the VIIIth nerve and lower brain stem. Lesions at these sites limit the ability to understand speech drastically. One may observe, for example, cases of VIIIth nerve tumor with a very poor speech discrimination score in the face of only slight reduction in the audiogram."

In our patient series, this phenomenon is most clearly exemplified by patient R. S. who obtained an articulation score of 36 per cent although showing only mild reduction of thresholds for pure tones. This patient, following subsequent radiologic treatment, achieved a substantially normal pure-tone audiogram; his discrimination score improved to 80 per cent, but was still lower than ordinarily expected with a normal audiogram. Other patients demonstrating results consistent with the "bottleneck principle" (see Fig. 1) are I. R. who scored 56 per cent, K. B. who scored 0, and probably S. W. with whom completion of standardized articulation testing was impossible but who clearly demonstrated profound reduction of speech discrimination in the affected ear.

There was, however, a second group of patients who demonstrated reduction of speech discrimination which was not necessarily inordinate in the light of the pure-tone audiogram (see Fig. 1). These were patients R. H. who scored 50 per cent, D. P. who scored 72 per cent and G. J. who also scored 72 per cent.

One patient, G. S., was of special interest. Since he was a Filipino and had limited facility in English and dialectic speech, standardized speech discrimination testing was diffi-

cult to perform. Using a standard children's P. B. list, with relatively strict scoring, however, a score of 86 per cent was obtained on the affected ear. It is conceivable that his actual discrimination facility was better than this score indicated, since marked differences between the unaffected and affected ears were not observed. Surgical findings on this patient were significant in that the tumor apparently arose from the Vth nerve, but was impinging upon the VIIIth nerve. The lack of reduced speech discrimination may be accountable on this basis. This patient represents the population of cerebellopontine angle tumors designated as Group II by Dix and Hallpike,[5] about which they observe, "Since the VIIIth nerve is not primarily involved, the VIIIth nerve signs are less in evidence than the neurological signs. . . . Indeed it is our experience in these tumors that some such unusual balance between otological and neurological abnormalities often provides a useful key to their diagnosis."

In discussing the varying auditory manifestations of cochlear and retrocochlear lesions, Jerger proposed a second principle: "the subtlety principle." This principle states, "The subtlety of the auditory manifestation increases as the site of the lesion progresses from peripheral to central." The principle suggests that end-organ lesions (with the exception of Ménière's disease) produce auditory disorders most notably characterized by depression of pure-tone thresholds and only secondarily affected by the loss of facility in the more demanding auditory tasks, such as speech discrimination. At the opposite end of the auditory system, cerebral lesions usually effect no reduction in sensitivity for pure tones and frequently incur no reduction in speech discrimination scores when obtained by standard testing procedures. Increasing the difficulty of the discrimination task by distorting the stimulus words will, however, demonstrate significant differences in discrimination facility between the affected and unaffected sides. Lesions at levels midway in the auditory system, notably at the cerebellopontine angle and the brain stem, produce some reduction in pure-tone thresholds but more marked deficits in the subtler task of speech discrimination.

Our findings may raise questions regarding the universality of this principle. Patient R. C. achieved a discrimination score of 12 per cent, patient I. W. scored 10 per cent, and patient E. S. scored 0 in the left ear and 34 per cent in the right ear. Although each of these patients probably demonstrated an undue reduction in discrimination as compared with the configuration of the audiometric curve (illustrating again the "bottleneck principle"), there was also a marked reduction in sensitivity for pure tones. We reiterate that six patients demonstrated complete absence of sensitivity for both pure tones and speech on the affected side. We believe that these findings may suggest that the application of the "subtlety principle" to patients with cerebellopontine angle tumors may be valid chiefly during the early stages of impairment. Referring again to the earlier reviewed initial test results obtained on patient K. B., it might be postulated that the initial auditory impairment resulting from tumors at the cerebellopontine angle may be as subtle as the pattern Jerger ascribes to patients with cerebral lesions but that the subtlety of impairment decreases as the tumor progresses.

Our observations of patients in this series, therefore, lead us to conclude that, although inordinate reduction of discrimination is often present, it can not be considered inevitable. In some instances fair and relatively consistent speech discrimination ability appears to be maintained, at least during the early stages of the symptom progression. In other instances, severe reduction in both pure-tone thresholds and speech discrimination scores occurs.

RESULTS OF TESTS FOR LOUDNESS RECRUITMENT.

In routine clinical practice, tests designed to demonstrate the presence or absence of disturbances in the perception of the loudness of stimuli are commonly employed when the site of the lesion producing reduced sensitivity for pure tones is in question. Until very recently, the loudness balance procedures originally proposed by Fowler have been the techniques most widely utilized to demonstrate disturbances in the loudness function. Major factors which appear to have accounted for this dependence on loudness balance tests are relative ease

of administration; the rapidity with which they can be accomplished with those patients where fatigability and anxiety limit the length of the testing session; the comparative simplicity of the judgments demanded of the patient, and the fact that special, often expensive equipment is not required.

Theoretically, recruitment is considered to be the result of cochlear dysfunction. As the result of a retrocochlear lesion, the auditory deficits attending cerebellopontine angle tumors should, therefore, not include disturbances of loudness functions. When applied clinically, however, loudness balance tests do not always yield all-or-none results. Dix and Hood,[4] based on results of applications of the Fowler loudness balance procedure, report that in 14 of 20 cases with cerebellopontine angle tumors, no evidence of recruitment was revealed; but that in the remaining six cases, recruitment was present but incomplete. In a later report, Dix[3] observes that full loudness recruitment was present in 8 per cent of the patients with cerebellopontine angle tumors, whom he had studied. He attributes the presence of recruitment to interferences to the cochlear blood supply incurred by the tumor. He cites as examples two patients who demonstrated complete recruitment preoperatively who later showed no recruitment postoperatively, although their thresholds for pure tones remained essentially the same.

Among those patients in our series with whom the administration of loudness balance testing was possible, the majority achieved results consistent with theoretical expectations, demonstrating no evidence of recruitment. Two patients, however, demonstrated findings suggestive of partial recruitment, and one patient demonstrated complete recruitment at one of the frequencies tested.

Patient J. C. (see Fig. 7) demonstrated what might be considered partial recruitment at 500, 1000, and 2000 cps. Note that at 500 cps. loudness increments totalling 90 db in the unaffected ear are matched by loudness increments totalling 75 db in the affected ear; at 1000 cps. increments of 90 db are matched with increments of 65 db; and at 4000 cps. increments of 90 db are matched with increments of 50 db.

Equivocal findings such as these, while suggestive of some aberrance in the loudness function, are difficult to interpret definitively.

Patient D. P. (see Fig. 7) demonstrated differing results at different frequencies. A pattern approaching complete recruitment by binaural loudness balance testing was demon-

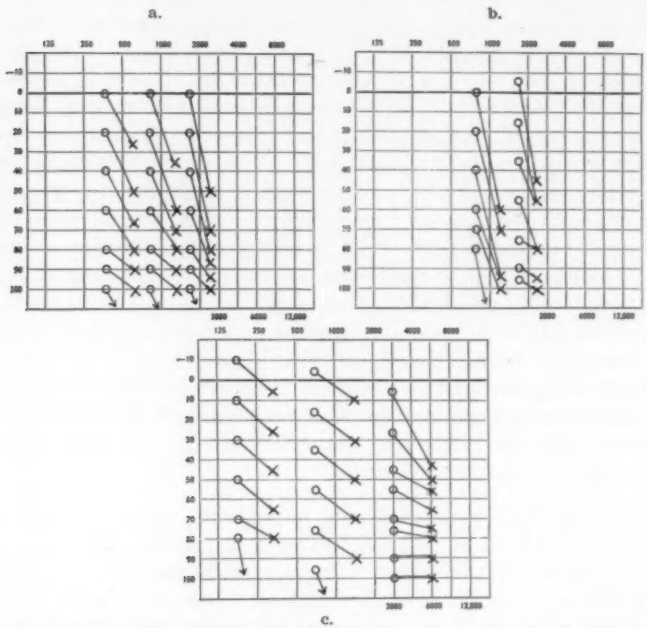

Fig. 7. Relatively atypical results of binaural balance tests conducted on three patients with cerebellopontine angle tumors. a.—Patient J. C. b.—Patient D. P. c.—Patient I. R.

strated at 2000 cps. although no recruitment was demonstrated at 1000 cps. Although no relationship can be established, it is interesting to note that this patient had the unusual trough-type curve. Patient I. R. (see Fig. 7) also demonstrated inter-frequency differences in binaural loudness balance testing. While there was no evidence of recruitment in the low and mid-frequencies, a pattern of complete recruitment was evi-

dent at 4000 cps. Since relatively good hearing for lower frequency tones was maintained, the differences between ears in the lower frequencies were not sufficient to make the patient an ideal subject for loudness balance testing at these frequencies. We cannot be absolutely certain, therefore, that a "no recruitment" pattern would have existed had interaural differences been greater. At the same time, the recruitment demonstrated at 4000 cps. may well be attributable to a pre-existing cochlear impairment at that frequency.

In general, our experience tends to reaffirm the value of the loudness balance technique for this particular patient population. As stated previously, the expected absence of recruitment was readily demonstrated with the majority of these patients. The phenomenon termed "over" or "hyper"-recruitment, so often seen in patients with labyrinthine disorders, was not evident with any of these patients. In the three instances where incomplete or complete recruitment was present, the results were sufficiently equivocal that the possible existence of a retrocochlear lesion remained under serious consideration. Our findings underline the importance of conducting loudness balance tests at as many frequencies as possible; since, in at least two instances, spurious observations would have been made, had only a single frequency been selected for study.

We do not find irrefutable evidence to support the need for the development of improved techniques for judging the presence or absence of loudness recruitment in this patient population. Nevertheless, we believe that the research which is striving to evaluate recruitment on a more quantitative basis offers the possibility of approaches which will be of considerable clinical value, especially in those instances where loudness balance techniques yield equivocal findings. We hope, however, that researchers will bear in mind the clinical feasibility of new measuring techniques. In clinical practice these techniques are frequently most crucial in instances where patients are least able to cooperate in highly demanding listening tasks.

The current literature makes frequent references to the

use of tone decay testing as a diagnostic tool in the identification of acoustic neoplasms. Regrettably, tests of tone decay have been added only relatively recently to the test battery routinely employed in our clinic for patients with suspected tumors. As a consequence, data on this testing procedure are not available for the majority of the patients reported here. The fragmentary information accrued to date indicates the possibility of somewhat greater variability of performance than other authors have reported; however, we would prefer to reserve comment until we have obtained information on a larger number of patients.

SUMMARY.

Reduced monaural auditory acuity is almost inevitably considered to be the most common and earliest symptom of cerebellopontine angle tumors. A patient's awareness of anacusis or dysacusis may frequently provide the initial impetus for seeking professional attention. It is, therefore, essential that the otologist and audiologist be aware of the early auditory manifestations of these tumors if they are to be identified during the early stages of development.

When the presence of a cerebellopontine angle tumor is in question, the audiologist may provide valuable assistance in the diagnostic process. Since the symptomatology may often be extremely similar to that of other pathologies, the consequent clarification of the auditory symptom as either a likely or unlikely manifestation of a cerebellopontine angle tumor can be of essential significance to the medical diagnostician.

Based upon the audiologic results obtained on 22 patients with subsequently surgically confirmed cerebellopontine angle tumors, we presently believe that it is possible to hypothesize a pure-tone audiometric pattern which may be characteristic of this problem. Although significant differences are observed, we believe these differences may be the result of variations in the point in the course of tumor progression at which the audiograms are obtained. An audiometric pattern which is unique to this patient population alone cannot be described; nevertheless, we believe that the patterns observed,

particularly during the early stages of the symptom progression, are sufficiently unusual to warrant careful investigation and close follow-up, especially when accompanied by subjective impressions of distortion of sound stimuli in the affected ear, headaches, and/or vertigo.

The inordinate reduction of speech discrimination scores as compared with pure-tone findings, currently described as one major auditory characteristic of these patients. was observed to be present with certain of the patients in this series; however, sufficient variation was observed to question seriously the inevitability of this finding, particularly in those patients where the auditory symptom had progressed beyond the initial, mild degree of impairment of sensitivity for pure tones.

On the basis of loudness balance tests, apparent absence of loudness recruitment was readily demonstrated in most of the patients on whom these tests could be conducted. In three instances the procedure yielded somewhat equivocal results. Although our observations tend to reaffirm the value of the loudness balance procedure, enough equivocation was evident to reaffirm the need for additional, simply administered loudness recruitment test procedures.

Our experience with these patients leads us to conclude that no single test in the standard battery offers infallible evidence of the existence of a cerebellopontine angle tumor, but that observing the results of as many measures of auditory function as can be appropriately administered, usually delimits the pathologies which can conceivably be present. There appears to be an unquestionable need for the refinement and standardization of testing techniques which are commonly used and for the development of new, more precise, procedures to assist in the identification of these tumors. In view of the practical problems which the audiologist frequently faces in the use of complex procedures with these patients, it is essential that clinical feasibility be considered as new tests are proposed.

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P. H. H.

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